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PREVENTION AND TREATMENT OF CARDIAC ARREST

Julian Johnson, M.D.
and
Charles K. Kirby, M.D., Philadelphia

There is nothing more catastrophic from the standpoint of the family, the surgeon, the anesthetist, and all other persons involved than a sudden unexpected death in the operating room. This is especially true if the patient is a healthy person who is undergoing some relatively minor elective operative procedure. This catastrophe occurs so infrequently in each hospital that the experience of any one surgeon or anesthetist in dealing with the problem may be small. For this reason, it is perhaps worth while to stress again the important points in its prevention and treatment.

When the circulation of the blood to the brain stops, it must be started again within approximately four minutes if the patient is to survive. This time limit is of the greatest importance. Experience has shown that, in the presence of this emergency, virtually no surgeon or anesthetist is able to act quickly enough unless he has previously gone through the problem in his mind and planned the steps to be taken. It is important, therefore, for every anesthetist and every surgeon, in whatever specialty, to familiarize himself with the essential points concerning the diagnosis and treatment so that, if he is suddenly brought face to face with it, he will not be hopelessly immobilized by the enormity of the catastrophe. The exact incidence of cardiac arrest is not known. It does occur, however, at least once or twice a year on most active surgical services. Our own figures suggest that it occurs about once in every 7,500 anesthetizations. We are interested in the treatment of cardiac arrest because, by quick and premeditated action, a life may be saved that otherwise would be lost. However, we are even more interested in the prevention of cardiac arrest and hope thereby to decrease the incidence of this catastrophe.

ETIOLOGY

When one sets aside the problem of cardiac surgery, the causes of cardiac arrest for all practical purposes may be placed in the following categories: Reflex phenomena, an overdose of or sensitivity to an anesthetic agent, hypoxia, or a combination of these factors. In the first group, due to reflex phenomena (vago-vagal reflexes), if there is not too great a delay, the heart will start up with a normal rhythm, as a rule, after only a few moments of compression. In the second group (too much anesthesia), one has the simple problem of maintaining an artificial circulation and respiration until the excess anesthetic agent is washed out of the system. The heart will then resume its normal activity. In the third group, when hypoxia is the primary cause of cardiac arrest, the problem is complicated by the fact that the brain and heart may have suffered considerable damage before the period of arrest actually began. The safe period for the cessation of the circulation would be reduced accordingly. In many instances it has been difficult to state definitely the cause of cardiac arrest, in which event everyone is apt to blame it upon the vago-vagal reflex. There is experimental and clinical evidence to suggest, however, that the vago-vagal reflex may be much more active in the presence of hypoxia. An example of this is the healthy man who "fights the anesthesia" and is cyanotic as the endotracheal tube is passed. Cardiac arrest has been observed frequently under such circumstances.

PROPHYLAXIS

If the incidence of cardiac arrest is to be materially reduced, there must be maximum cooperation between the surgeon and anesthetist. The surgeon should not push the anesthetist too far in demanding deep anesthesia, and the anesthetist should remember that his objective is to maintain the highest possible arterial oxygen saturation and a normal pH of the blood. All agree that, if the anesthetic agents are carefully chosen and are administered in safe doses and if, above all, hypoxia and hypercapnia are avoided by adequate ventilation, this catastrophe should rarely occur. Cardiac arrest is not limited to poor-risk patients but occurs very frequently and perhaps most often in good-risk patients. This suggests that, since no difficulty is anticipated in such patients, the greatest

From the Harrison Department of Surgical Research, Schools of Medicine, University of Pennsylvania, and the Surgical Department of the Hospital of the University of Pennsylvania.
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possible care is not exercised in their management. The fact that the margin of safety is so great in these persons may lead both the anesthetist and surgeon to rely too much upon that fact. The patient who is a recognized poor risk from the cardiac standpoint has a narrow margin of safety. It is especially important in such patients for the anesthetist to avoid hypoxia and hypercapnia. The surgeon is responsible for prophylactic considerations directly related to the operative procedure. Safeguards against sudden hemorrhage should be employed during the dissection. Strong traction on thoracic and abdominal viscera should be avoided.

Operations upon the heart constitute a separate category. Ventricular fibrillation is the great fear. The direct insult to the myocardium has been thought to be the inciting cause of the ventricular fibrillation. We formerly, as did many others, administered procaine hydrochloride or procaine amide (Pronestyl) hydrochloride intravenously in an effort to decrease the myocardial irritability. More recently we have abandoned that practice because of the lack of evidence of its usefulness. It is thought that the results have been just as good, if not better, since that time. We have come more and more to believe that the most important thing in the prevention of ventricular fibrillation is the maintenance of maximum oxygenation of the myocardium. It has been found that greater than normal ventilation may be required in patients undergoing heart surgery in order to keep the myocardium pink. Time and again the myocardium has been found to be slightly cyanotic with the usual amount of ventilation, but, when the ventilation is increased, the color of the myocardium may improve immediately. We feel that the securing of maximum oxygenation of the myocardium before direct trauma to it is the most important protective measure against ventricular fibrillation. The surgeon should stop and allow the anesthetist to make a conscious effort to obtain maximum oxygenation and optimal color of the myocardium, especially before cutting into either ventricular wall. We still employ procaine locally when entering the ventricles and believe it to be useful. Obviously the heart should be manipulated as gently and briefly as possible.

DIAGNOSIS

The early diagnosis of cardiac arrest is the most important single factor in its treatment, as we have previously stressed.[1] For the most part this must rest in the hands of the anesthetist. Since the patient can survive only about four minutes of a cessation of circulation, it goes without saying that valuable time may be lost if the anesthetist doesn't keep a constant observation of the patient. If the pulse is felt only every five minutes, it would obviously be possible for the circulation to have stopped for more than four minutes before it was first detected. It must be the objective of the anesthetist to avoid cardiac arrest but, if it should occur, to recognize that fact immediately. When the anesthetist announces that the pulse and blood pressure have suddenly disappeared, it must be assumed that the circulation has ceased. This is no time for consultation or meditation. The circulation must be started again. The desire to confirm the diagnosis of cardiac arrest before instituting treatment is understandable. However, the usual methods of determining whether the heart is beating are too time consuming and unreliable. If the surgeon is operating in the chest, he can immediately see or feel the heart. If he is operating in the abdomen, he can feel the heart through the diaphragm. If he is in the region of a large artery, he can feel that. On the other hand, time spent looking for a stethoscope to listen for the heartbeat or time spent with an ophthalmoscope trying to see whether blood is flowing through the retinal vessels may cost the patient his life. When the anesthetist reports that the pulse and blood pressure have suddenly and unexpectedly disappeared and the surgeon is not already in a position to feel the heart or a large artery, the left side of the chest should be opened in order to ascertain the true state of affairs immediately. Under such circumstances we have taken the point of view that to expose the heart is a diagnostic procedure.

TREATMENT

If the heart is to be exposed quickly, it is important that some thought be given to the problem before it arises. It would be nice to have such equipment as sterile instruments and drapes, but their absence should not cost the patient his life. All one really needs is a knife. A pair of gloves may be put on if they are immediately available. Given a knife, the surgeon should have his hand on the heart in about 15 seconds. The incision should be made in the left fourth or fifth interspace and carried down to the pleura; the pleura should be opened with the handle of the knife. Once the pleura is open, the underlying lung can be protected by a finger as the rest of the interspace is opened widely. The right hand can then be forced between the ribs and the heart palpated. If it is in asystole, it will be still. If it is in ventricular fibrillation, it may give the sensation of fine muscular twitchings. It is not important at this point to make such a differential diagnosis. If the heart is not beating forcefully, it should be compressed rhythmically with the right hand. The costal cartilage above and below may be cut with the knife in the left hand as intermittent cardiac compression is being carried on with the right hand. This will decrease the pressure of the ribs on the hand or wrist. A rib spreader will be helpful when available.

A small heart may be compressed satisfactorily with one hand by holding it between the thumb and fingers. When dealing with a larger heart, it is often better to compress it between the two hands. Some surgeons prefer to open the pericardium in order to compress the heart more efficiently. We prefer to remain outside the pericardium but to free the mediastinum anteriorly enough to get the hand partially around the heart. When the pericardium is opened, care should be taken not to penetrate the cardiac wall with the tips of the fingers or thumbs. The effectiveness of the cardiac massage may be judged by the production of a good pulse and bleeding from the wound. The vessels may be caught with hemostats when available. The rate of the cardiac compression should be as rapid as can be conveniently maintained. A rate of about 60 per minute is recommended, for it is difficult to maintain a faster rate. If two or more opera-

1. Johnson, J., and Kirby, C. K.: Cardiac Resuscitation, S. Clin. North America **29**: 1745, 1949; Surgical Treatment of Ventricular Fibrillation, Ann. Surg. **134**: 672, 1951; Cardiac Resuscitation, J. Mt. Sinai Hosp. **18**: 141, 1951.

tors are available to work in shifts, a rate of 80 or more may be maintained. Our experiments have suggested that more blood is moved by the more rapid rate and that it is not profitable to allow a longer interval between compressions in order to allow the heart to fill.[2] If the heart seems empty, the blood volume should be increased by giving fluids intravenously at a fairly rapid rate. Caution must be observed, however, not to overload the circulation.

While the surgeon is producing artificial circulation, the anesthetist must induce respiration artificially. Mouth-to-mouth breathing may be used until proper equipment is available. An anesthesia machine with a face mask, using 100% oxygen, is the best method and should be used as soon as it is possible. Once the patient is well oxygenated, time may be taken to insert an endotracheal tube. It is the objective of the cardiac massage by the surgeon and the artificial ventilation by the anesthetist to produce adequate oxygenation of the blood and tissues of the entire body. If the patient's color returns to normal and there is a good palpable pulse, an adequate artificial circulation is probably being produced. If the heart does not start beating normally after a short period of adequate oxygenation, ventricular fibrillation should be suspected.

VENTRICULAR FIBRILLATION

The irregular movements of the myocardium felt on palpation are pathognomonic of ventricular fibrillation. When the fibrillary twitchings involve very small muscular segments, it may be necessary to visualize the myocardium to be certain of the diagnosis. In any event, the color of the heart must be returned to normal by means of adequate artificial circulation before efforts are made to defibrillate the heart. Occasionally a heart in ventricular fibrillation will revert to normal rhythm spontaneously. This may also occur after the use of procaine or procaine amide hydrochloride intravenously. The most reliable method of defibrillating the ventricles, however, is by electric shock.[3] The shock throws all the muscle fibers into a powerful contraction simultaneously, causing the heart to go into standstill thereafter. The ventricles are then apt to resume contraction with a normal rhythm.

The electric device used by us to defibrillate the ventricles is easily assembled. Actually, the current taken directly from the house line would probably be successful in most instances. An isolation transformer is placed in the circuit to decrease the risk to the surgeon. A variable resistance is used so that with a small heart, especially in children, the current (by the short circuit test) may be reduced to about 2 amperes. In the normal adult heart, the resistance must be almost or entirely removed from the circuit. With a very large heart, the regular house current (110 volts) may not be powerful enough to defibrillate the ventricles. We have, therefore, inserted a variac in our experimental defibrillator that will produce up to 270 volts. There is a very real danger, however, of producing burns of the myocardium with such high voltages, and routine use is not recommended. A smaller voltage (125 to 135 volts) should be used for routine purposes. The duration of the current is also important, especially when using high voltages, if burns are to be avoided. In recent experiments in our laboratory, it has been shown that 220 volts for 1/10 second will defibrillate hearts under conditions when 110 volts for 1/10 second will not.[4] In clinical practice, however, when 220 to 270 volts have been required to defibrillate the ventricles, the patients have not survived, possibly because of the circumstances requiring such high voltages. The size and character of the electrodes and the firmness with which they are pressed against the ventricles are also important variables in the amount of current actually delivered to the myocardium.

The surgeon should take every precaution to avoid an electric shock to himself or to the anesthetist. Time is available to take all possible precautions, since there is no rush to defibrillate the heart, as long as an adequate respiration and circulation are being maintained. The isolation transformer greatly reduces the possibility of the surgeon receiving a dangerous shock. By wearing two pairs of gloves and having insulated handles on the electrodes, the risk of a shock should be reduced to a minimum. The surgeon should also stand on a wooden stool and take care not to touch the operating table as the shock is applied. The anesthetist should remove his hands from the patient and the table as the current is applied. The current should be applied for less than 0.5 second when a single shock is used. For a series of shocks, the duration of each shock should be very brief. If the patient's entire body does not contract, the current is probably not strong enough. If the heart is not defibrillated upon the first attempt, repeated attempts should be made after obtaining maximum oxygenation of the myocardium.

RESULTS

During the seven year period from Jan. 1, 1946, to Jan. 1, 1953, there were approximately 95,000 anesthesias given at the Hospital of the University of Pennsylvania. Of these, about 70,000 were spinal or general anesthesias. If one discounts the operating room deaths due to such things as an uncontrollable hemorrhage and to operations directly upon the heart, there were 13 instances of cardiac standstill or ventricular fibrillation. These instances represent unexpected catastrophies. This would give an incidence of about 1 in 7,500 anesthesias. In addition, there have been six patients in whom ventricular fibrillation developed during direct attacks on the heart, a problem present only in hospitals where cardiac surgery is done. In previous publications, attempts at cardiac resuscitation upon patients suddenly dying in our wards were included in the data. With the enthusiasm of the resident staff for cardiac massage, this number has no statistical significance. There have been no successes in this group. It is not urged that all such efforts be abandoned, for a number of hearts have been started again only to fail later, but it is suggested that the circumstances under which pa-

2. Johnson, J., and Kirby, C. K.: Experimental Study of Cardiac Massage, Surgery **26**: 472, 1949.

3. Wiggers, C. J.: Cardiac Massage Followed by Countershock in Revival of Mammalian Ventricles from Fibrillation Due to Coronary Occlusion, Am. J. Physiol. **116**: 161, 1936. Beck, C. S., and Mautz, F. R.: Control of Heart Beat by Surgeon, with Special Reference to Ventrical Fibrillation Occurring During Operation, Ann. Surg. **106**: 525, 1937.

4. Kirby, C. K.; Johnson, J.; Engelberg, J., and Rovis, R.: Ventricular Fibrillation: Experimental Study, to be published.

tients die in the wards and the delay in instituting treatment, in general, mitigate against success. In the 13 instances of totally unexpected cardiac arrest in the operating room, 7 patients (including one with ventricular fibrillation) have been successfully resuscitated. In the six cases of ventricular fibrillation occurring during direct attacks on the heart, all hearts were defibrillated but only three of these patients survived.

Attempted Cardiac Resuscitation in the Operating Room

	1946	1947	1948	1949	1950	1951	1952	Total
Excluding Cardiac Cases								
Attempts	1	2	1	2	2	3	2	13
Successes	0	1	0	2	2	1	1	7
All Cases								
Attempts	1	2	1	3	4	4	4	19
Successes	0	1	0	3	4	1	1	10

In 1949 and 1950 we were greatly encouraged by 100% success in the operating room. This excellent record has not been maintained in 1951 and 1952. Three of the six failures in these two years were in persons with very large hearts undergoing cardiac surgery when ventricular fibrillation developed. Shock of 220 to 270 volts was required to defibrillate the ventricles. It may be that the duration of the current was too long. At any rate, two of the patients exhibited obvious myocardial burns at autopsy. In the third, the heart reverted to fibrillation several times, and the patient died in the operating room. The other three patients might have been saved. There was too great a delay in two, and in one, a 74-year-old patient, the myocardium was badly torn during cardiac massage, the pericardium having been opened.

SUMMARY

It is suggested that every surgeon and anesthetist familiarize himself with the essential steps to be taken in the event of sudden unexpected cardiac arrest in the operating room. Once the circulation has stopped, it must be started again within about four minutes if the patient is to survive. When the surgeon is not in a position to feel the heart or a large artery, he should be prepared to expose the heart through the left side of the chest if the anesthetist announces that the blood pressure and pulse have suddenly and unexpectedly disappeared. If there is not too great a delay in instituting an adequate artificial circulation and respiration, the heart should resume a normal rhythm in a short time. If ventricular fibrillation is present, it is most effectively dealt with by electric shock, after the myocardium has been adequately oxygenated.

3400 Spruce St. (4) (Dr. Johnson).

CARDIAC DISABILITY AND DEATH CAUSED BY STRAIN: PROBLEM IN WORKMEN'S COMPENSATION

Louis H. Sigler, M.D., Brooklyn

There is much controversy on the causal relationship between physical and emotional strain and cardiac disability and death. Some believe that strain seldom produces cardiac disease. Others believe that it is a frequent cause of cardiac disease and death. In disability due to coronary disease, for instance, Master and co-workers [1] found that only 2% of 530 persons with coronary thrombosis had the attack during unusual exertion and 37% more during mild activity. They therefore conclude that an attack occurring after strain is merely coincidence. On the other hand, in 950 autopsy cases of all forms of coronary sclerosis in the armed services, Yater and co-workers [2] found that, in the younger age group, the terminal cardiac attack occurred during strenuous activity in as many as 32% and during mild and moderate activity in 52% more. In only 15% did it occur at rest. In 100 cases of fatal coronary sclerosis in young soldiers, reported by French and Dock,[3] 35% died one to several hours after vigorous exercise. Thus, statistics vary with the sampling of the population and with the definition of coronary disease and so they cannot be offered as proof.

Emotional and physical strain may produce disability and death in any form of cardiac disease, such as rheumatic, syphilitic, or hypertensive; discussion here will cover only the coronary atherosclerotic form of heart diseases because many patients here may show no symptoms or signs of heart disease until acute manifestations develop. From a viewpoint of cardiac disability, we must consider coronary atherosclerosis as a single entity disease. The use of such terms as coronary insufficiency, coronary failure, coronary thrombosis, myocardial infarction, atypical myocardial infarction and subendocardial myocardial necrosis [4] in describing the various manifestations of the disease give us only limited descriptions. At any given time, the clinical and electrocardiographic expressions of the disease depend only on the location, degree, duration, and rapidity of onset of the myocardial ischemia and the myocardial damage that the underlying coronary disease has produced in the patient; this idea was propounded by Hecht.[5] If no myocardial ischemia is produced, because of insufficient narrowing of the lumen or because of the presence of a good collateral circulation, the person may be symptom free, carry on normal activities, and the electrocardiogram may remain normal for many years.

If myocardial ischemia develops only on strain, excitement, or activity and subsides immediately after, the

1. Master, A. M.; Dack, S., and Jaffe, H. L.: Factors and Events Associated with Onset of Coronary Artery Thrombosis, J. A. M. A. **109**: 546 (Aug. 21) 1937.
2. Yater, W. M.; Walsh, P. P.; Stapleton, J. F., and Clark, M. L.: Comparison of Clinical and Pathologic Aspects of Coronary Artery Disease in Men of Various Age Groups: Study of 950 Autopsied Cases from the Armed Forces Institute of Pathology, Ann. Int. Med. **34**: 352, 1951.
3. French, A. J., and Dock, W.: Fatal Coronary Arteriosclerosis in Young Soldiers, J. A. M. A. **124**: 1233 (April 29) 1944.
4. Littman, D., and Barr, J. H., Jr.: Acute Atypical Coronary Artery Insufficiency, Circulation **5**: 189, 1952.
5. Hecht, H. H.: Concepts of Myocardial Ischemia, Arch. Int. Med. **84**: 711 (Nov.) 1949.

anginal syndrome will occur only while these factors are operative. The electrocardiogram, likewise, may show some abnormalities only during the ischemic phase. If local myocardial ischemia develops spontaneously or after effort and does not subside after rest, the anginal syndrome is severer, lasts much longer, and recurs more frequently. If the ischemia is prolonged and severe, focal necrosis of the heart muscle will occur, and the absorption of necrotic material may produce variable elevation in temperature, acceleration of the blood sedimentation rate, increase in the number of white blood cells, increase in the heart rate, and electrocardiographic abnormalities. The severity of these changes will depend upon the degree of necrosis. If the acute myocardial ischemia is extreme, circulatory collapse may occur due to cardiac asystole, ventricular fibrillation, or reflex peripheral vascular failure, and death may result before there is time for myocardial infarction to develop. If the patient survives, the subjective manifestations may continue for hours or days, and objective findings of massive infarction such as changes in the character of the heart sounds, gallop rhythm, tachycardia, drop in blood pressure, pulmonary congestion, and rise in temperature may develop. There may also be a marked acceleration of the blood sedimentation rate, a greater or lesser increase in the white cell count, and characteristic electrocardiographic findings of gross infarction.

Looking at the disease from this broad aspect prevents the erroneous view that disability and death here may be due only to one particular pathological process such as coronary thrombosis. In Yater's series [2] of 950 autopsies, deaths from coronary disease were observed to have occurred in cases with or without coronary occlusion and with or without myocardial infarction. Furthermore, occlusion was found to be due to sclerosis alone in 39% of cases, to thrombosis alone in 23%, and to combined sclerosis and thrombosis in 25%. In 13% of cases that showed gross myocardial infarction, no occlusion was found. The occurrence of myocardial infarction without coronary occlusion and even without significant coronary sclerosis has been observed before. Gross and Sternberg [6] have reported 15 cases of the latter. The infarction here may be due to severe prolonged deprivation of the blood supply to the myocardium caused by coronary vasospasm, tachycardia, shock, severe anemia, polycythemia, and possible humoral elements such as excessive secretion of epinephrine or other vasopressor substances. Dack and co-workers [7] have observed 7 cases of subendocardial myocardial infarction in 40 fatal cases of pulmonary embolization. They attributed it to general anoxemia and reflex coronary vasoconstriction. Shock was also undoubtedly a factor.

Physical and/or emotional strain is a potent factor in precipitating these various acute manifestations of coronary disease; this has been demonstrated before by Paterson,[8] Horn and Finkelstein,[9] Nelson,[10] Blumgart,[11] Graybiel,[12] and others. Physical strain increases the aortic and, thus, the intracoronary pressure transiently and may produce acute changes in the coronary vessels in cases where there is marked atheromatosis. The main effect of emotional strain is to stimulate the sympathetic nervous system, producing the various tachycardias, arrhythmias, and cardiac overactivity, making greater demands on the coronary blood supply. It also produces general vasoconstriction and rise in pressure, which is transmitted to the coronary system. Sympathetic stimulation of the adrenal glands and the accompanying increase in epinephrine secretion further speeds the heart rate and raises the blood pressure. Waters and deSuto-Nagy [13] have demonstrated experimentally that epinephrine injection also produces necrotizing changes of the coronary arteries.

In many cases an acute coronary process is caused by intimal hemorrhage, often brought about by strain. Paterson [8] observed that capillary outgrowth occurs directly from the lumen of the coronary arteries in areas of intimal thickening. The thinness, fragility, and lack of support of the capillary walls and their exposure to the direct intracoronary blood pressure predisposes them to rupture. The accumulation of blood in the intima acutely accelerates the atherosclerotic process by the deposition of formed substances that induce reactive tissue response, acute degeneration, and extension of the atheromatous plaque toward the lumen of the coronary artery. Coronary thrombosis also appears to be caused in many cases by these hemorrhages, which induce disruption and dissolution of the intima, liberating thromboplastic material. Although transient, recurring rise in intracoronary pressure may thus be the main mechanism in the pathogenesis of acute coronary changes in most cases, a marked drop in pressure such as occurs in shock sometimes appears to be the underlying cause of coronary insufficiency, coronary thrombosis, and myocardial infarction. Blumgart and co-workers [14] observed 11 cases of multiple small coronary occlusions and 38 cases of single occlusions due to shock. They attributed the pathological changes to a fall in blood pressure, to blood concentration, and to tachycardia occurring in shock.

The occurrence of cardiac injury due to physical and/or emotional strain in the presence of coronary atherosclerosis is more frequent than is generally suspected. I have collected over 250 cases where an acute cardiac insult was definitely traceable to such strain. Many of these had no workmen's compensation claims, and the reliability of the history was unquestionable, as in the following example: A newly wed woman, 30 years old, while on her honeymoon participated in a strenuous handball game for 10 minutes; immediately after this, severe pain developed in the anterior portion of her chest, radiating to the back and left arm, accompanied by cold, clammy

6. Gross, H., and Sternberg, W. H.: Myocardial Infarction Without Significant Lesions of Coronary Arteries, Arch. Int. Med. **64**: 249 (Aug.) 1939.

7. Dack, S.; Stone, J.; Paley, D. H., and Master, A. M.: Pulmonary Embolism: Precipitating Factor of Acute Coronary Insufficiency, Dis. Chest **15**: 72, 1949.

8. Paterson, J. C.: Factors in Production of Coronary Artery Disease, Circulation **6**: 732, 1952.

9. Horn, H., and Finkelstein, L. E.: Arteriosclerosis of Coronary Arteries and Mechanism of Their Occlusion, Am. Heart J. **19**: 655, 1940.

10. Nelson, M. G.: Intimal Coronary Artery Hemorrhage as Factor in Causation of Coronary Occlusion, J. Path. & Bact. **53**: 105, 1941.

11. Blumgart, H. L.: Relation of Effort to Attacks of Acute Myocardial Infarction, J. A. M. A. **128**: 775 (July 14) 1945.

12. Graybiel, A.: Diseases of the Heart: Review of Significant Contributions Made During 1942, Arch. Int. Med. **71**: 713 (May) 1943.

13. Waters, L. L., and deSuto-Nagy, G. I.: Lesions of Coronary Arteries and Great Vessels of Dog Following Injection of Adrenalin: Their Prevention by Dibenamine, Science **111**: 634, 1950.

14. Blumgart, H. L.; Schlesinger, M. J., and Zoll, P. W.: Multiple Fresh Coronary Occlusions in Patients with Antecedent Shock, Arch. Int. Med. **68**: 181 (Aug.) 1941.

perspiration, and a sensation of extreme weakness and fainting. She was removed to a hospital where examination disclosed evidence of posterior left ventricular wall infarction.

Among other cases that I have observed, the attack occurred during or immediately after an exciting argument or a competitive card game, during an exciting ball game, after mowing a lawn, during sexual intercourse, or other similar activities described before.[15] However, we must appreciate the fact that, by the time an attack occurs under strain, coronary atheromatosis in one or more branches has progressed to a stage where complete shutting off of the blood supply to a given portion of the heart may even occur spontaneously, without any known strain. For this reason the determination of causal relationship between strain and cardiac injury in any case is often difficult, especially in workmen's compensation cases where the reliability of the history may be questionable. I will attempt to present, however, certain criteria, illustrated by case reports, that may help us to decide if causal relationship exists between a given strain and a resulting cardiac injury.

Before discussing these criteria we must clarify the meaning of the term "cardiac injury" as determined clinically. By this term we mean the occurrence of more or less acute clinical manifestations and laboratory evidences of heart disease, following strain, which were not present before the strain. If heart disease was known to have been present before, we must have proof that an acute exacerbation, clearly precipitated by the strain, occurred. Furthermore, the clinical manifestations of heart disease following the strain must be of prolonged nature, not transient, and the damage must be clearly demonstrable. Thus, the occurrence of the anginal syndrome following strain that immediately subsides at rest does not constitute causal relationship of the strain and cardiac injury. It merely points to transient myocardial ischemia due to coronary insufficiency induced by the strain in the presence of underlying coronary disease and calls for the avoidance of such strain in the future. In other words, it is a warning sign that myocardial injury may ultimately result if the particular strain causing the anginal syndrome is continued.

The strain that brings about an acute attack must be very severe and must be followed immediately by the clinical signs of a cardiac insult to constitute causal relationship. It need not necessarily be an unusual type of strain, for, in the progressive course of coronary disease, a vulnerable stage is sooner or later reached when even a usual strain, provided it is of severe enough nature, may produce injury. Following is an example: A 51-year-old foreman in a clothing factory, who had never experienced any disturbances referable to his heart, had an argument with another worker about poor workmanship and then helped lift a machine weighing about 150 lb. (68 kg.). He immediately experienced severe pain in the precordium with cold sweat, nausea, vomiting, and collapse. He was removed to a hospital where a diagnosis of anterior left ventricular wall infarction was made. The close association of the attack with the excitement and the lifting of the heavy load in this case leaves no doubt of causal relationship. That he had before occasionally resorted to somewhat similar activities without cardiac injury is no argument against causal relationship. In the first place we must assume that the preexisting coronary disease was not advanced far enough before to be vulnerable. Secondly, we have no definite measure by which to gauge the exact amount of mental and physical effort employed at any given time in accomplishing the same work.

A major acute cardiac insult may be considered causally related to strain even if the onset of symptoms is not very severe and dramatic, provided the symptoms develop immediately after the strain and continue over a long period. The presence of the injury to the heart in these cases may occasionally be overlooked. The following is an example: A 59-year-old electrician was wiring a new building and had to pull four wires through a conduit, a distance of about 75 ft. (23 m.). The conduit had four bends at angles of 90 degrees each. To prevent the wires from getting stuck, he had to use strong jerks. He immediately felt a choking sensation in the precordium and the throat, with a "sense of weight" over his chest, which was followed 15 minutes later by severe pain in both shoulders. He rested one hour and was taken home because he could not continue working. Throughout the rest of the day he had a sensation "like gas" in the chest, which was partly relieved by rhubarb and soda. He returned to work the following day and continued working for six days, periodically experiencing "troubled breathing" and some of the sensations noted above. He was finally examined by a doctor, who took an electrocardiogram that revealed anterior left ventricular wall infarction. Intimal capillary hemorrhage induced by the strain undoubtedly caused acute changes in a preexisting atheromatosis in this case, with progressive occlusion and infarction over a period of days.

Injury to the heart caused by strain may be of temporary nature, and recovery is possible. This is true especially where no extensive myocardial infarction occurred. If recurrence of myocardial injury takes place at a later time while the person is engaged in usual, nonstrenuous work, the recurrence can not be attributed to the strain that caused the original injury. This is illustrated by the following case: A man 58 years old, who never had symptoms referable to his heart, had to push a hand truck containing tools weighing 150 to 200 lb. (68 to 90 kg.) up a ramp at an inclination of 30 degrees. When he reached the top of the ramp, a height of about 30 ft. (9.2 m.), he suddenly felt severe pain across the anterior portion of his chest, which radiated into the left arm and left hand. The pain lasted about one-half hour and recurred on many occasions during the rest of the day, with freedom from pain for about one-quarter to one-half hour between the attacks. He was sent home from work, and, after he rested through the following day, the pain subsided. He then returned to work, and the pain recurred again on many occasions. He was finally removed to a hospital where he remained two weeks. Several electrocardiograms revealed an acute ischemic pattern and possible necrosis of the left ventricle. He experienced pain intermittently for about six weeks, but with continued rest it gradually diminished, so that about

15. Sigler, L. H.: Cardiovascular Disease: Fundamentals, Differential Diagnosis, Prognosis and Treatment, New York, Grune & Stratton, Inc., 1949.

seven months after the onset the pain had entirely subsided, and he returned to work. An electrocardiogram obtained at this time was within normal limits. He worked for six months thereafter at usual, nonstrenuous work without any discomfort whatsoever. At the end of that time, and while continuing his usual work, he again began to have recurring attacks of pain, and electrocardiographic evidence of myocardial ischemia again developed.

It is evident here that the patient had marked atherosclerosis of the coronary system and that an acute coronary insult resulting in myocardial ischemia and possible necrotizing changes was induced on the first occasion by the severe strain. At prolonged rest, a sufficient collateral circulation developed with clinical recovery. The subsequent attacks were in the nature of spontaneous onset of acute coronary insufficiency, due to the progress of the coronary disease, and had no relation to the original strain.

The question whether prolonged and repeated strain under unhealthy conditions may bring about cardiac injury, even though such strain may be of a usual kind and not of a sudden acute nature, cannot be definitely answered. Nevertheless, if a person who was apparently asymptomatic before subjecting himself to such strain showed symptoms and signs of cardiac injury during the performance of his work and such symptoms and signs persisted, it is reasonable to claim causal relationship. Following is an example: A seaman, 31 years old, was at sea on board a gasoline transport freighter. During the entire trip he had to do arduous cleaning of large empty gasoline tanks and chipping off of old paint with a heavy hammer. The air in the tank had a heavy gasoline odor, and his sleeping quarters had poor ventilation. While doing his work he experienced a peculiar intermittent oppression in his chest, and at night he was awakened from his sleep with severe precordial pain lasting one hour. The pain recurred periodically the rest of the trip. When he reached New York, a doctor prescribed glyceryl trinitrate (nitroglycerin), which gave him relief. On his return ocean trip the pain persist, and when he reached shore he was hospitalized. Repeated electrocardiograms revealed an ischemic pattern with necrosis localized in the posterior wall of the left ventricle. After seven weeks of hospital care, he had complete symptomatic relief, and the electrocardiogram showed only residual damage. He returned to lighter work and has had no recurrence of symptoms. We must assume here that the strenuous activities together with improper hygienic conditions produced in this case acute coronary changes with myocardial ischemia in the presence of latent, asymptomatic, preexisting coronary disease.

As was stated before, an acute cardiac insult frequently occurs apparently spontaneously. In many such cases, the attack may be traced to overeating, to reflex disturbances from other organs, and to other similar causes. From a viewpoint of workmen's compensation, attacks traced to these sources, even though occurring while at work, are, of course, not compensable. Nevertheless, strenuous work performed at the same time that the other factors are operative may be contributory, and, therefore, compensability should perhaps be considered in some cases where it is now regarded only as a definite additional causative factor. The physician therefore often encounters a difficult problem in rendering an opinion in some of these cases. Following is an example: A volunteer fireman, 43 years old, went with his company on a half-mile march that lasted about one hour. He had no subjective complaints before, during, or soon after the march. He and his group then indulged in eating considerable amounts of food, consisting of frankfurters and sauerkraut on rolls, and drinking a great deal of soda and beer. He joined in the singing and playing. After one-half hour severe chest pain developed, and he fell to the ground in an unconscious state and in shock. He was removed to a hospital and died five days later.

Autopsy revealed some cardiac enlargement, softening of the anterior wall of the left ventricle, some degenerative changes of the myocardial fibers, atherosclerosis of the ascending aorta with distortion of both coronary ostiums, intimal coronary sclerosis with pinpoint narrowing of the lumen of the proximal 3 cm. of the left coronary artery. At a compensation hearing the question arose as to whether or not the march of one-half mile was the precipitating cause of the acute cardiac insult. Inasmuch as there were no symptoms referable to the heart either during or soon after the completion of the march, it sounded logical to assume that the march had no effect on the heart. On the other hand, the excitement, overeating, and copious drinking for a half hour after the march, with their reflex effect on the heart, could explain the onset of the acute condition.

In most cases, the physician's opinion as to causal relationship between strain and an acute cardiac insult depends largely upon the reliability of the history in the case. Following is an example: A 49-year-old man, a plumber, stated that he has been perfectly well before the incident in question. He was hanging a wash basin weighing about 40 lb. (18 kg.). He claimed that the basin slipped, and, to prevent it from falling to the floor, he had to lean forward with great force to grasp it. He immediately had a feeling of constriction in the precordium, which lasted several hours and was associated with nausea and cold sweat. He was taken to a hospital where he remained six weeks. During his stay in the hospital, there was no demonstrable clinical evidence of gross myocardial infarction. Repeated electrocardiograms showed an ischemic pattern. He continued experiencing recurring precordial pain on any exertion for many months after he left the hospital. It developed at the compensation hearing that he had been subject to recurring spontaneous attacks of chest pain for a long time before the work episode, but the one he experienced at his work was more severe.

From the fact that the patient had recurring attacks of coronary insufficiency for a long time before the particular work incidence and that no changes other than ischemia were noted in the heart, we may assume that the work was not the underlying cause of the onset of ischemia. That it might have temporarily accentuated the process is a possibility. However, a temporary accentuation is different than an initiation of a process from a compensation viewpoint. The mere lifting of a 40 lb. basin and holding it against the wall, discounting his claim that he suddenly had to use "great force" to

prevent it from falling, is not sufficient strain to initiate an attack of an acute cardiac insult. Attacks of myocardial ischemia, necrosis, and gross infarction may be followed by complete recovery, partial disability, or total disability. The outcome in any case depends mainly upon the degree of residual damage, the severity of the underlying coronary disease, the frequency of recurrence of acute insults, and, to a great extent, upon the early recognition and proper management of the individual case. The survival period even after acute myocardial infarction may often be very long if there are no recurrences. In a series of cases I reported,[16] there was one patient who lived as long as 35 years. The average, of course, was far lower.

The amount of activity a person can carry on after recovery from an attack may be amazing. Lewis [17] reported on a 43-year-old man who, on recovery from posterior wall infarction, went skiing on many occasions at high altitudes and carried on a great deal of other activities for five and a half years after the attack. I have observed many patients suffering from the effects of myocardial infarction who have carried on a great deal of activity for many years after recovery as in the following example: A 43-year-old shipping clerk, with the driver of a truck, lifted several linoleum rolls, club chairs, and sofas from the ground into the truck. He immediately experienced severe pain across the anterior portion of the chest that was accentuated as he continued working and was soon followed by marked weakness, cold perspiration, and pallor. The pain recurred with greater or less severity on his way home, and, 35 minutes after he arrived home it became very excruciating and lasted all through the night. An electrocardiogram two days later revealed anterior left ventricular wall infarction. After two months of rest, the electrocardiogram returned to normal, and he made complete symptomatic recovery except for slight precordial discomfort on marked exertion. It is now seven years since the attack, during which time he has continued working without discomfort, performing practically the same type of heavy work he had done before. The electrocardiogram also has remained normal.

In a follow-up of 412 cases of coronary occlusion and 141 cases of coronary insufficiency for five years after the attack, Master and Jaffe [18] found that 83% of these patients were employed, with 60% working full time. My experience in this area has shown approximately the same percentages. Partial and even total disability may follow an acute cardiac insult in many cases. The causes may be (1) paroxysmal attacks of severe tachycardias or arrhythmias originating from an irritable focus in the heart induced by the injury; (2) recurring myocardial ischemia and infarction; (3) severe anginal attacks; (4) congestive failure; and (5) emotional disturbances that are often associated with fear that death may result from work. In some cases more than one of these factors are operative.

16. Sigler, L. H.: Prognosis of Angina Pectoris and Coronary Occlusion: Follow-Up of 1,700 Cases, J. A. M. A. **146**: 998 (July 14) 1951.

17. Lewis, W. H., Jr.: Coronary Occlusion: Report of Unusual Activities After Recovery, J. A. M. A. **114**: 484 (Feb. 10) 1940.

18. Master, A. F., and Jaffe, H. L.: Complete Functional Recovery After Coronary Occlusion and Insufficiency, J. A. M. A. **147**: 1721 (Dec. 29) 1951.

It is often difficult to determine the extent of residual cardiac disability. Where the amount of remaining damage is marked, and objective manifestations of heart disease such as congestive failure and recurring severe tachycardias and arrhythmias are evident, we can easily determine the extent of disability. Even in the presence of the anginal syndrome alone, which is mainly a subjective manifestation, we can often tell if the manifestation is real and serious by associated visible phenomena that the patient exhibits. In some cases, however, it is hard to tell if the complaints are real or feigned as in the following example: A man, 59 years old, lifted with another man a barrel of cement weighing about 200 lb. (90 kg.) and carried it a distance of about 5 ft. (1.5 m.). He immediately experienced severe pain in the left anterior precordium and left arm down to the fingers, and he lost consciousness. He was removed to a hospital where evidence of posterior left ventricular wall infarction was found. For a period of five years following his discharge from the hospital he claimed to have recurring precordial pain and attacks of dizziness. From time to time he appeared to have lost consciousness and to have convulsive-like movements and marked tachypnea. On many occasions he was picked up on the street during these attacks and was removed to different hospitals only to be soon discharged because of lack of abnormal physical findings. The electrocardiogram was persistently stable, showing merely the remains of the old infarction. Furthermore, he lived on the fourth floor of a walk-up building and was observed to go up and down the four flights several times a day without discomfort. I had the occasion to observe him during one of his attacks. It consisted of induced spasms of various muscle groups and tachypnea with attempt at fainting. He showed no evidence of brain irritation or of a hyperactive carotid sinus reflex. There were also no changes in the heart rate or rhythm during the attack, ruling out possible transient heart block with the Adams-Stokes syndrome. That myocardial infarction developed after strain and that he was thus entitled to disability compensation, there is no quesetion. It is questionable, however, if the disability should have been of such long duration, because the symptoms he exhibited could not be attributable to heart disease or its sequelae. The duration of disability following cardiac injury due to strain varies in different cases and depends upon many factors, the most important being the functional capacity of the heart. Each case must be studied individually before arriving at an opinion. The ultimate prognosis in any case depends largely upon the early recognition of the underlying pathological changes and the proper medical management of the individual case. Contrary to the usual present day practice of early ambulation, it has been my experience that, in serious acute myocardial damage, long bed rest followed by prolonged relative rest is essential in the prevention of irreparable permanent damage. The extent and duration of rest should be prescribed carefully according to the needs of each individual patient.

SUMMARY

Physical and emotional strain may be definite exciting factors in the pathogenesis of acute coronary changes and myocardial injury in the presence of coronary

atherosclerosis. Inasmuch as an acute insult frequently occurs spontaneously, however, the causal relationship between a given strain and the resulting myocardial injury is often open to controversy. Such relationship should be considered established when a severe strain is followed by acute cardiac symptoms and definitely demonstrable acute myocardial damage. This is true even if similar strain has produced no ill effects before when the coronary atherosclerosis was not "ripe" for an acute insult. An honest history of the events leading to an attack is most essential in establishing causal relationship.

The incidence of clinical recovery from an acute myocardial insult and the rate of continued employment is high. If another acute coronary insult with myocardial injury occurs in the course of usual activity after complete clinical recovery from an acute insult due to strain, the second attack can not be attributed to the original strain, which brought about the first attack. It should be considered merely a spontaneous insult in the course of progressive coronary degeneration. If the second attack occurs after unusual strain, the cardiac injury should be attributed to the second rather than the first attack. Partial or total permanent disability may often follow a cardiac injury due to strain. The extent of disability of each person should be gauged primarily by the degree of underlying coronary disease, the extent of residual permanent myocardial damage, and the functional capacity of the heart.

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EXPERIENCE WITH THE MECHANICAL HEART

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It has been approximately three years since the mechanical heart project was started. The apparatus is so devised that four different types of substitutions for the heart and lungs can be performed. These consist of (*a*) bypass of the right side of the heart, (*b*) bypass of the left side of the heart, (*c*) bypass of both sides of the heart while the lungs continue to perform their functions, and (*d*) bypass of the heart and lungs while the mechanical oxygenator performs lung function. A detailed description of the apparatus has already been published.[1]

There were many problems to be solved during the early work. These chiefly concerned the problems of clotting, hemolysis, contamination, and uncontrollable oozing. The trouble that was encountered with clotting has been largely eliminated by giving the proper dosage of heparin and by coating the mechanical apparatus with silicone, which greatly tends to lessen the clotting process. Much work was done in the experimental engineering laboratory to construct the type of pump that is the least injurious to the blood. The apparatus that was selected seemed to handle the blood with the least destruction. Since the apparatus is constructed so that all parts that contact the blood are sterilized by ordinary autoclaving, the problem of infection has not been significant. In a total of over 100 animal experiments, there was not one instance of blood stream infection after extracorporeal circulation. The problem of oozing during an operation in which a large amount of heparin is used is still a formidable problem. There need not be excessive loss of blood, however, if the bleeding points are carefully ligated. Most of these procedures required considerable time, and it was not necessary to give protamine solution at the conclusion of the operation, since the effect of the heparin had begun to wear off.

BYPASS OF RIGHT SIDE OF HEART

Several investigators have successfully bypassed the right side of the heart in animals. Leeds, Puciss, and Siegel [2] as well as Sewell and Glenn [3] were among the first to report substitutions for the right side of the heart. With a bypass of the right side or a bypass of the entire heart, there are certain hypotensive reflexes present that may be severe, especially if some degree of anoxia exists. The systemic blood pressure is partially regulated by this reflex mechanism on the right side. As the atrium and great veins become distended, nervous impulses traveling through the vagus nerve are sent upward to the brain and cause an acceleration of the heart rate and an increase in the systemic pressure. Conversely, as the atrium and great veins are emptied and the pressure in them is diminished, a nervous impulse is initiated that causes a slowing of the heart and a fall in the systemic blood pressure. If significant anoxia exists, this hypotension may be severe. It is imperative, therefore, that good oxygenation be maintained during the procedure.

In a bypass of the right side of the heart, the blood is drawn from the right atrium and passed through the right side of the mechanical heart and back into the branch of the pulmonary artery supplying the right lower lobe of the lung. A cannula is placed in the artery to the lower lobe so that it points centrally, and in this manner blood is supplied to the middle lobe, the right upper lobe, and the entire left lung. Figure 1 illustrates the blood flow in the bypass of the right side. The right ventricle is thereby completely bypassed. The pulmonary artery proximal to its bifurcation then can be totally occluded. This prevents backflow from the pulmonary circuit and permits ex-

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The artificial heart mechanism reported here was made by the Research Laboratories Division, General Motors Corporation.

1. Dodrill, F. D.; Hill, E., and Gerisch, R. A.: Some Physiologic Aspects of Artificial Heart Problem, J. Thoracic Surg. 24: 134, 1952.

2. Leeds, S. E.; Puciss, I., and Siegel, P.: Shunting of Blood from Right Heart by Means of Pump with Operation on Interior of Heart, in Surgical Forum of the American College of Surgeons, 1950, edited by O. H. Wangensteen and others, Philadelphia, W. B. Saunders Co., 1951, p. 265.

3. Sewell, W. H., Jr., and Glenn, W. W. L.: Experimental Cardiac Surgery: II. Further Observations on Development of Perfusion Pump for Purpose of Shunting Venous Return Directly into Pulmonary Artery, in Surgical Forum of the American College of Surgeons, 1950, edited by O. H. Wangensteen and others, Philadelphia, W. B. Saunders Co., 1951, p. 265.

posure of the pulmonary valve. The blood also may be taken from the venae cavae, and clamps may be placed across the cavae between the cannulas and the atrium so that no caval blood enters the atrium. If this method is used, a separate plan for handling the coronary sinus blood must be used. In addition, there is considerable flow from the thebesian system into the right heart that also must be handled separately.

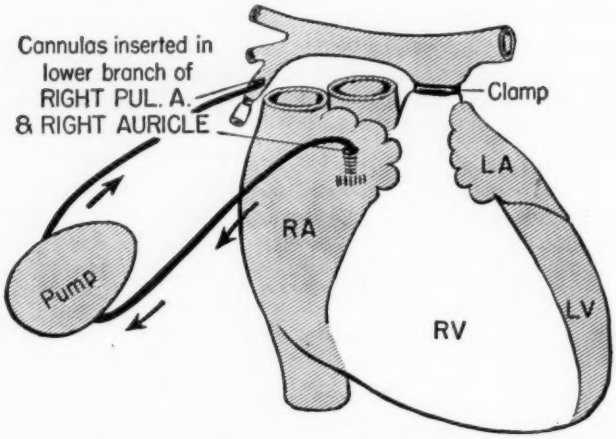

Fig. 1.—Diagram showing method of bypass of the right side of the heart.

There are not many diseases of the heart in which a bypass of the right side is a practical procedure. Obviously both the interventricular and interatrial septums must be intact for this procedure. Diseases of the pulmonary and tricuspid valves are examples. Unfortunately the majority of patients with pulmonary valve obstruction have other defects, particularly defects of the septums that preclude a right bypass. Some patients with congenital pulmonary valve stenosis, however, do not have such concomitant defects and the right bypass is a practical procedure in these patients. In patients with congenital pulmonary stenosis, a defect in either of the cardiac septums is manifested by some lessening of oxygen saturation and concomitant polycythemia. Moreover, if a significant right to left shunt exists, it may be demonstrated by angiocardiography; therefore, if no lack of arterial oxygen saturation exists and if angiocardiography fails to demonstrate a right to left shunt, it is probable that none exists. It should be borne in mind, however, that, with a defect in the septum and with a pressure in the right ventricle that equals that in the left, there is no shunting of blood from right to left, although the actual defect exists.

A 16-year-old boy with congenital pulmonary stenosis and apparently without a defect of the septums was successfully operated on on Oct. 21, 1952. This patient was large for his age, weighing 178 lb. (80.7 kg.). The history included congenital heart disease since infancy. During the past several years, there was much disability from dyspnea on slight exertion. There was a loud harsh systolic murmur over the pulmonary valve area that completely replaced the heart sounds. The blood pressure was 110/70 mm. Hg. The electrocardiogram showed marked right ventricular hypertrophy and strain. The thoracic roentgenogram showed enlargement of the right side, with diminished vascularity of the lung fields. On fluoroscopic examination of the thorax, no pulsations were seen in the arterial branches, although the main pulmonary arteries were greatly dilated. Cardiac catheterization showed that the right ventricle pressure was 190/0 mm. Hg. The pulmonary artery could not be entered at this catheterization. The arterial oxygen saturation was 90 vol. %. The red blood cell count was 4,950,000 per cubic millimeter. The remainder of the examination was essentially normal. Since this patient had a high ventricular pressure and since there was no evidence of polycythemia, clubbing, or a right to left shunt, it was felt that a defect of the interventricular or interatrial septum did not exist. The arterial oxygen saturation of 90 vol. % could be explained on the basis of low pulmonary artery pressure.

The heart was exposed through a transverse incision in the anterior thorax. The third intercostal spaces on both sides were incised; the sternum was transected; and both pleural cavities were opened widely. This gave a complete exposure of the heart. The preoperative diagnosis was substantiated by the presence of a vigorous systolic thrill at the base of the pulmonary artery as well as the marked right-sided enlargement. The right atrium was huge. The main pulmonary artery was greatly dilated. The right atrium was cannulated with a large cannula and another cannula was placed in the pulmonary artery to the right lower lobe and pointed centrally so that the blood could flow to the middle lobe, right upper lobe, and the entire left lung. The distal portion of the artery to the lower lobe was occluded. The mechanical apparatus was ready to be connected and to take over the

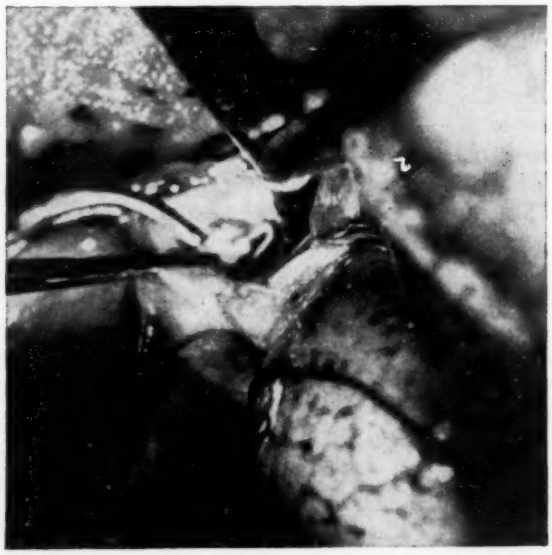

Fig. 2.—Photograph showing stenotic pulmonary valve at operation in 16-year-old boy with congenital pulmonary stenosis.

function of the right ventricle. With the patient under anesthesia, a Cournand cardiac catheter was placed in the right ulnar artery and passed upward into the innominate artery. This was connected to a direct writing apparatus so that the arterial pressure could be recorded continuously. A continuous electrocardiogram was recorded. These two records were carefully observed by the cardiologist during the operation. The patient was

given 5 mg. of heparin per kilogram of body weight, and to the amount of blood necessary to fill the artificial mechanism was added 1 mg. of heparin per 100 cc. of blood. The mechanical heart was then slowly turned on until the right ventricle was completely bypassed. At this time, the thrill at the base of the pulmonary artery was no longer present, indicating that blood was not being ejected from the right ventricle. A clamp was then placed across the main pulmonary artery proximal to its bifurca-

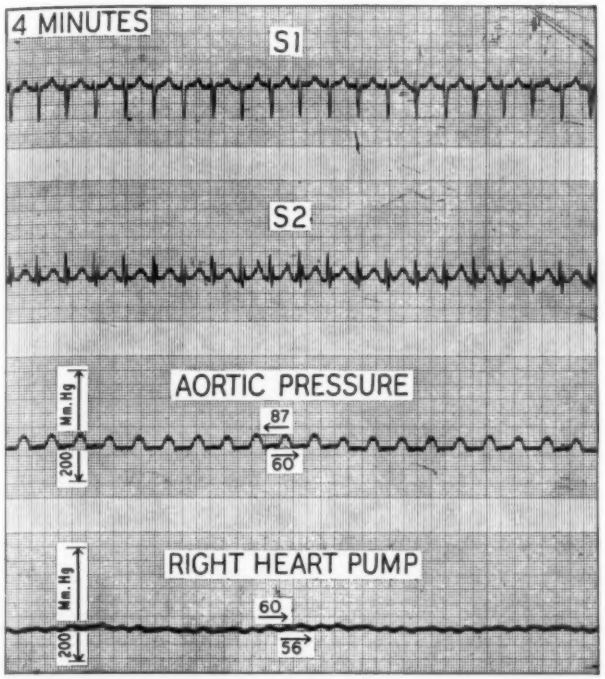

Fig. 3.—Electrocardiograms, aortic pressure, and pressure in the right side of the mechanical heart during bypass of right side of the heart in a 16-year-old boy.

tion. Another light, noncrushing clamp was placed across the pulmonary conus, and the pulmonary valve was completely isolated from the circulation. A transverse incision was then made in the base of the pulmonary artery, and the stenotic pulmonary valve was readily exposed. It was markedly stenotic and approximately 5 mm. in diameter. Figure 2 shows the stenotic pulmonary valve in this patient at operation. A valvuloplasty under direct vision was carried out. The pulmonary artery was totally occluded for a period of 25 minutes while the mechanical heart completely took over the function of the right ventricle. During this time, the left side of the heart and the lungs continued to perform their functions; the patient remained in apparently good condition. At the completion of the valvuloplasty, the incision in the base of the pulmonary artery was closed; the clamps were then removed; and the mechanical heart was slowly shut off, allowing the right ventricle to resume its former function. Figure 3 is a record of the electrocardiogram and arterial pressure pattern during the time the pulmonary artery was clamped off. The canulas were removed. The artery to the right lower lobe was sutured, and the atrial appendage was ligated.

This patient made a good recovery, although during the first few postoperative days there was evidence of mild pulmonary edema demonstrated by roentgenologic examination as well as by some mild hemoptysis. It was not definitely known whether this pulmonary edema was due to the fact that the mechanical heart was supplying blood to the pulmonary circuit under a greater pressure than the lungs were accustomed to or whether a sudden and great enlargement of the pulmonary valve also tended to produce pulmonary edema. This cleared within a few days, and from that point the patient made an excellent recovery. The cardiac catheterization was repeated six months later when the pressure in the right ventricle was 50/0 mm. Hg. It is now seven months since the operation, and the patient continues to remain in excellent condition. He is no longer dyspneic and, in fact, is carrying on as a normal person.

BYPASS OF LEFT SIDE OF HEART

A bypass of the left side of the heart has been carried out experimentally by several investigators. Among the first to do this procedure were Sirak, Ellison, and Zollinger,[4] Kantrowitz and Kantrowitz,[5] Wesolowski and Welch,[6] and others. Extensive left-sided bypasses have been carried out in animals by myself and my associates. The work has reached a gratifying stage wherein the left side of the heart in dogs may be completely bypassed with opening of the left atrium and exposure of

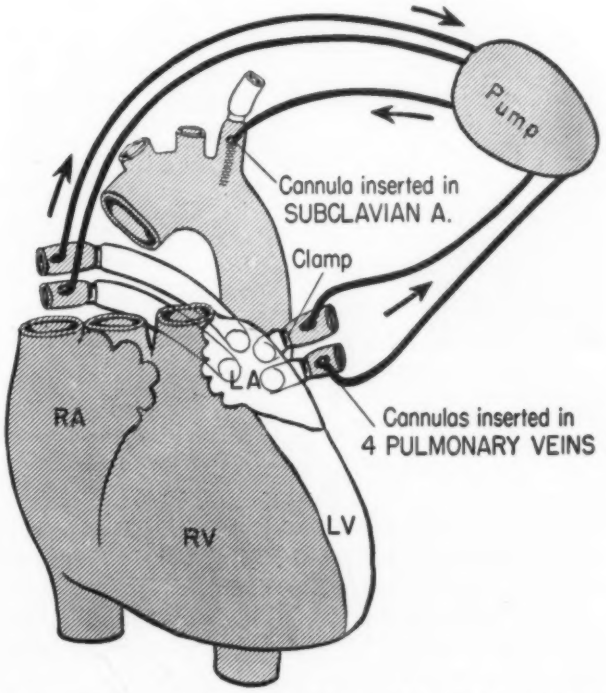

Fig. 4.—Diagram showing method of bypass of the left side of the heart.

the mitral valve with almost perfect results. During the last 15 or 16 consecutive experiments in dogs in which the left atrium and mitral valve were exposed, there was

4. Sirak, H. D.; Ellison, E. H., and Zollinger, R. M.: Cardiotomy into Empty Left Ventricle, Surgery **28**: 225, 1950.

5. Kantrowitz, A., and Kantrowitz, A.: Observations on Use of Experimental Artificial Left Heart, in Surgical Forum of the American College of Surgeons, 1950, edited by O. H. Wangensteen and others, Philadelphia, W. B. Saunders Co., 1951, p. 265.

6. Wesolowski, S. A., and Welch, C. S.: Pump Mechanism for Artificial Maintenance of Circulation, in Surgical Forum of the American College of Surgeons, 1950, edited by O. H. Wangensteen and others, Philadelphia, W. B. Saunders Co., 1951, p. 226.

not one death. In dogs, at least, the hypotensive reflex that may be initiated on the right side is seldom elicited. Blood may be taken from the pulmonary veins and passed through the left side of the mechanical heart and back into the aorta by way of the left subclavian artery. The left atrium, the mitral valve, and the left ventricle are then free of blood. This procedure is obviously dependent on a competent aortic valve since, if the aortic valve is not competent, blood will flow back into the ventricle and thence into the atrium. This would prevent exposure of the structures. For that matter, a competent aortic valve is necessary for either the left-sided bypass or the complete heart and lung bypass. Figure 4 illustrates the direction of blood flow in this procedure.

A 50-year-old woman with rheumatic mitral valve disease was operated on by means of the left-sided mechanical bypass. This patient had attacks of rheumatic fever in childhood and known rheumatic mitral valve disease for the past several years. She became increasingly incapacitated. There was a moderate diastolic murmur

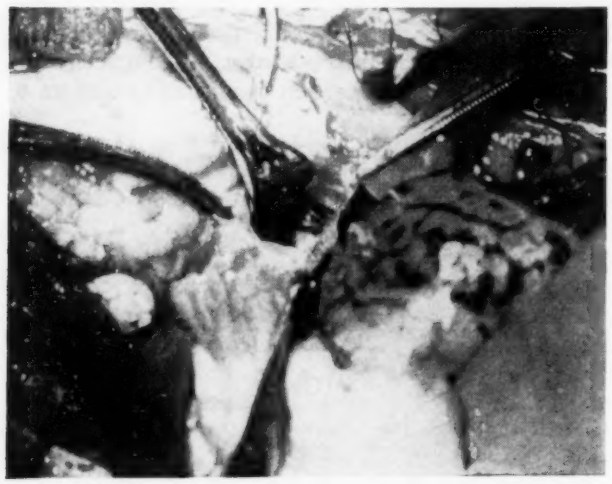

Fig. 5.—Photograph of empty left atrium showing the edge of the mitral valve in a 50-year-old woman with mitral valve disease during bypass of left side of the heart.

present at the apex. The heart was regular in rhythm, and the blood pressure was 120/70 mm. Hg. The electrocardiogram showed right axis deviation, with right ventricular hypertrophy. The roentgenogram examination of the thorax revealed enlargement of the right side. Cardiac catheterization showed an increase in the right ventricle pressure to 45/0 mm. Hg. Pulmonary artery pressure was approximately 42-45/8 mm. Hg.

The heart was exposed through an anterior transverse thoracic incision in the third intercostal space. The pulmonary veins were cannulated, and blood was taken from this point and passed through the left side of the mechanical heart and back into the aorta by way of the subclavian artery. Clamps were placed on the veins between the cannulas and the atrium. After the mechanical heart took over the function of the left ventricle, the arterial pressure pattern, which was being recorded, showed the usual sign wave of curve indicating that the left ventricle was not ejecting blood into the aorta. The atrium was then collapsed and the appendage was opened; this revealed a dry atrium. The mitral valve could be seen and was enlarged. Figure 5 shows the empty left atrium and also a portion of the mitral valve. The atrium was then sutured, the clamps between the cannula and the atrial wall were removed, and the mechanical heart was gradually shut off. The left ventricle took over its former function, and the arterial pressure pattern was the typical arterial pattern. No particular attempt was made to evacuate the air in the left side of the heart. As the mechanical heart was gradually turned off, some blood flowed into the atrium and into the ventricle and displaced the air upward to the highest point; this was probably aspirated into the mechanical heart and filtered out. At any rate, there has been no evidence of air embolism in our entire group of experimental animals. When the atrium and left ventricle are free of blood, they are only potential cavities and are not distended. This makes the volume of air present in them much less than would be suspected. The cannulas were removed from the pulmonary veins and the incisions in them were sutured with fine silk, reestablishing their continuity. The cannula was then removed from the subclavian artery, and the artery was ligated. The chest wall incision was closed, and two tubes were inserted intercostally for drainage. The patient made a fairly satisfactory recovery, although about 36 hours postoperatively there was some evidence of cardiac failure with some peripheral edema. This responded to medical treatment. It has now been four months since the operation, and the patient is doing well.

In addition to the left-sided bypass in which blood is taken from the pulmonary veins and passed into the aorta, another type of bypass of the left side of the heart has been carried out in which blood is taken from the atrium by cannulating the left superior vein and passed through the mechanical heart and back into the aorta. By this method, all the blood can be diverted from the left side of the heart, although the atrium cannot be opened and the structures in the interior of the heart exposed because the tip of the suction cannula would admit air. With the blood being completely diverted from the left side of the heart, however, prolonged and severe manipulations of the mitral valve have been carried out that were possible only if the systemic circulation was artificially maintained.

By this method, a 40-year-old man was operated on about a year ago. A left-sided bypass was carried out, although the heart was not opened. This patient's case was previously reported.[7] He had mitral regurgitation, and, after the mechanical heart had taken over the function of the left ventricle, a prolonged, blind manipulation of the mitral valve was carried out; this lasted for 17 minutes. During this time, the finger was inserted down through the mitral valve into the ventricle and the chordae tendineae were stretched to allow valve closure. This manipulative procedure is shown in figure 6. During this time, there was no hypotension and no arrhythmias. It is probable that such a severe procedure could not be carried out when the left ventricle is maintaining the systemic circulation. This patient has had great relief of his symptoms. Previous to the operation, the pulmonary capillary pressure was increased to approximately 18 mm. Hg. as determined by cardiac catheterization. Eight

7. Dodrill, F. D.; Hill, E., and Gerisch, R. A.: Temporary Mechanical Substitute for Left Ventricle in Man, J. A. M. A. **150**: 642 (Oct. 18) 1952.

months after the operation, cardiac catheterization was repeated and the pulmonary capillary pressure had fallen 7 to 8 mm. Hg, or essentially to normal.

Two other patients with rheumatic mitral disease were operated on in this way. The blood was drawn from the right atrium and passed back into the aorta by way of the subclavian artery. Both of these patients had had arterial emboli. As the left side of the mechanical heart

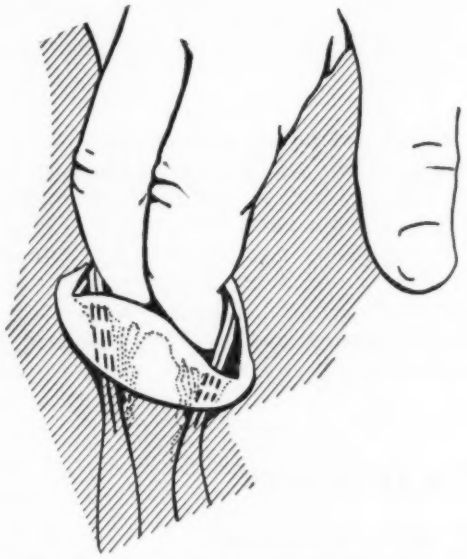

Fig. 6.—Diagram showing procedure for stretching the chordae tendineae in a 40-year-old man with mitral regurgitation.

was turned on, soft, congealed blood clots were withdrawn from the atrium and were filtered out in the mechanical heart. Although the mitral commissurotomy in these patients was done blindly, it is probable that had the soft clots not been aspirated from the atrium severe, if not fatal, complications might have ensued.

BYPASS OF BOTH SIDES OF HEART

Both sides of the heart may be bypassed by the mechanism. The two sides of the mechanical heart may be substituted for the two ventricles, while the lungs continue to perform their physiological function. One patient was operated on in whom such a substitution was carried out. This patient was an 18-year-old girl with congenital pulmonary stenosis in whom it was not definitely known whether a ventricular septal defect existed. The patient was markedly disabled; she was profoundly dyspneic on mild exertion. There was great right-sided enlargement of the heart shown by both physical examination and a thoracic roentgenogram. The electrocardiogram showed marked right ventricular enlargement. The pressure in the right ventricle at the time of cardiac catheterization was 178-180/0 mm. Hg. The heart was exposed through a transverse anterior thoracic incision in the third intercostal interspace with transection of the sternum. The right side of the mechanical heart was connected as for a right-sided bypass. The blood was taken from the right atrium and passed back into the artery to the lower lobe. The cannula was pointed centrally to supply the pulmonary circuit. On the left side, the left atrium was cannulated through its appendage, and the blood was put back into the aorta by way of the left subclavian artery. Figure 7 illustrates a bypass of both sides of the heart. The two sides of the mechanical heart were, therefore, simultaneously turned on, and the functions of both ventricles were taken over by the apparatus. The pulmonary valve was then exposed and enlarged under direct vision. Unfortunately, there also existed a profound infundibular stenosis in addition to the valve stenosis that could not be corrected. The mechanical heart completely maintained both sides of the circulation for 35 to 40 minutes while the valvuloplasty was carried out. Unfortunately, this patient died on the fourth postoperative day. Autopsy showed severe atelectasis of the left lung. During the postoperative period, there was evidence of some brain damage that occurred during the procedure because of either anoxia or cerebral embolization. The brain was not examined at the postmortem examination.

COMBINATION OF HYPOTHERMIA AND THE MECHANICAL HEART

Hypothermia in combination with the mechanical heart has considerable advantage. Under hypothermia, the circulation is slowed, the volume of blood being circulated is lessened, the heart rate slows, and the amount of blood being ejected by the coronary sinus is greatly diminished. Since the volume flow is reduced, it is not necessary to use the four major lobes of the lungs for their physiological function. Instead, one, or at the most two, of the lobes is sufficient for the circulating blood volume. This minimizes the amount of cannulation to be done. In the connection of the mechanical heart for a bilateral bypass, the blood flow through one lung is interrupted while the opposite lung maintains the physiological function completely. After one lung has been connected in the circuit, the opposite lung is removed from the circuit by occluding its inflow and outflow. By this

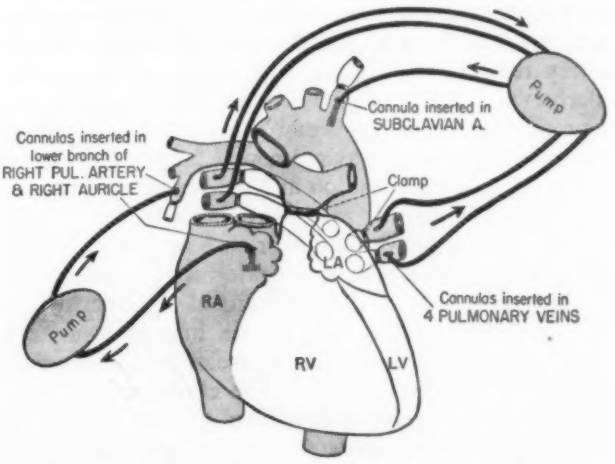

Fig. 7.—Diagram showing method of bypass of both sides of the heart.

means, one lung is used for its physiological function. This seems to be completely adequate, especially under moderate degrees of hypothermia.

COMMENT

Various ways of bypassing the heart need exploration. Of these various types of procedures, none are competitive except possibly the bypass of both ventricles and the complete bypass of the heart and lungs. During the

earlier days of work with the mechanical heart, it was felt by some that a unilateral bypass of the heart could not be carried out because the remaining side could not continue to perform its function. Apparently this is not true. It has been definitely shown by both prolonged animal experimentation and the results in living patients that the function of one or the other ventricles may be taken over by mechanical means while the other side and the lungs continue to perform their functions.

It is easier to connect the mechanical heart oxygenator to the anatomic structures than to do a unilateral bypass. With this procedure, blood is taken from the right side and passed through the mechanical heart oxygenator and back into the aorta. This required only two cannulations that are done on the right side. The connection of the mechanical apparatus to the anatomic structures in which the lungs are used for their functions is more difficult from the surgical standpoint. If the lungs can be used for their natural functions, however, it is probably more satisfactory than mechanical oxygenation. All of this work with the mechanical heart is in its infancy. The results demonstrated to date by the various investigators working in this field offer hope that eventually the heart may be bypassed for various types of intracardiac procedures. Blind procedures to correct intracardiac diseased valves represent a great step forward in surgery. Large numbers of patients have been relieved of their symptoms by these methods. It is probable, however, that a much more precise and exact corrective procedure can be performed on these deformed valves, if they can be exposed and the surgery done under direct vision. There is practically no surgical procedure carried out in any part of the body, aside from the heart, that is not under direct vision.

SUMMARY

A discussion of various ways of bypassing of the heart is presented. These consist of a bypass of the right side, the left side, and both sides of the heart, with the lungs for their natural functions, and a bypass of the heart and lungs. Four patients have been successfully operated on by one of these procedures. The pulmonary valve on the right side and the mitral valve on the left side have been successfully exposed and surgically corrected with survival of the patients.

OTOSCLEROSIS IN IDENTICAL TWINS

FACTORS IN ONSET, EXACERBATION, AND STABILIZATION

Edmund Prince Fowler, M.D., New York

In 1947 I published in detail my findings in a study of five pairs of identical twins with otosclerosis.[1] These data will not be repeated here, since the discussion in this paper will concern the etiology of otosclerosis. Heredity looms large in the picture of otosclerosis because it occurs more frequently in some families and generations than in others. However, although heredity is one factor and a very real factor, it is the sole cause neither of otosclerosis nor of any other dyscrasia. More properly it is a predisposition or abnormal sensitiveness to critical internal and external environmental factors that brings it into being. Predisposition and abnormal sensitiveness may be and commonly are inheritable and may operate by way of the soma or psyche or both in parents, in offspring, and in siblings in varying degrees for generations or forever. There is a constitutional "pecularity" or "susceptibility"—terms that in the human connote a more tangible link with certain disease conditions than they do in the lower animals. The clinician may occasionally detect susceptibility even in the absence of disease, and this he must strive to do if preventive treatment is to be of great value. Human heredity must be studied at a different level from genetics in laboratory animals. I failed to find histological otosclerosis in monkeys after they had been subjected to prolonged small and massive doses of the estrogens. In other animals, attempts to produce it by various means have met with failure. No one has produced it in any of the lower animals, probably because there is no heredity predisposition to the disease in any animal but man. In the lower animals, resistance to it seems to be absolute. In human beings, resistance varies; this is evident from records of incidence in siblings of large families with histories of the disease for generations. To what extent the differences in incidence are due to environmental conditions such as diet, endocrine imbalance, stress, shocks or emotional states, or to varying levels of susceptibility is not known. In some families, after decades of genetic distribution, all siblings for one or more generations may show no deafness from otosclerosis.

Identical twins are supposed to be genetically similar because both twins come from the same egg, but I have repeatedly found otosclerosis clinically limited to one twin or clinically severer or appearing some years earlier in one twin than in the other. Moreover, in one or both of a pair of identical twins, as in nontwins, one ear alone may reveal the presence of deafness due to otosclerosis. This occurs in about one-third of the twins I have examined early and also in nontwins. Guild's histopathological data revealed the incidence of otosclerosis (not the deafness) to be unilateral in roughly one-third of his material.[2] This would seem to indicate that, in spite of heredity, one ear escaped or possessed a partial resistance to otosclerosis for some time. It does not mean that the bone of the ear that showed no lesion, or the person from whom the skull bone was removed, did not possess through inheritance a susceptibility to otosclerosis in both ears. Nor does it mean that, if death had occurred

Read before the Section on Laryngology, Otology and Rhinology at the 102nd Annual Meeting of the American Medical Association, New York, June 4, 1953.

1. Fowler, E. P.: Otosclerosis in Identical Twins: Five Case Histories, Ann. Otol., Rhin. & Laryng. **56**: 368 (June) 1947.

2. Guild, S. R.: Histologic Otosclerosis, Ann. Otol. Rhin. & Laryng. **53**: 246 (June) 1944.

and the autopsy had been performed a few years later, both ears would not have shown the lesion. Both heredity and environment are frequently slow in their manifestations. It is interesting to note that unilateral otosclerosis was observed oftener in the younger than in the older patients I have examined. The two ears of any person might be considered identical twins, as they both come from the same egg and usually look much alike, and so, both in identical twins and "identical ears," we are provided with an opportunity for studying differences in internal and external environmental factors affecting one or the other or both.

OPPORTUNITIES FOR STUDY

Our knowledge concerning the inheritance of right and left side differences is confused, even our knowledge of the mechanism of right and left handedness. Variations in blood supply, whether anatomic, traumatic, or pathoautonomic, are possible factors in some persons. These factors may differ in any two persons, in fraternal twins, in identical twins, and, generally but not so obviously, in any one person's pair of ears. I call attention to these observations because they suggest several possibilities for research on our problems.

Recently isotopic tracer studies reveal that calcium is rapidly withdrawn from and redeposited in the bones of human beings as it is in animals. Human bone metabolism and its variations can now be measured. It would seem that at long last we are in a position to carry these studies on to confirmation by coordinated clinical and preclinical laboratory findings. In any event we should soon have some additional data for pertinent consideration. I anticipate that it will be possible to determine preclinically the changes in bone metabolism brought about by emotional stress. I believe that, in the presence of an inherited predisposition to otosclerosis, such changes in bone metabolism will be found to be at least one of the determining factors in the etiology of the lesion of otosclerosis and of its exacerbation and stabilizations.

The unique bone dyscrasia producing the typical losses of hearing observed in otosclerosis does not effect the hearing until the annular ligament of the stapes footplate is affected sufficiently to impede airborne sounds, or until the auditory neural elements become involved. The time lapse between the onset of the lesion of otosclerosis and the appearance of the outstanding symptoms (deafness) may be months or even years, depending on the location, multiplicity, rate and intermittency of growth, and the direction of extension of the lesion. If the rate of growth alone could be estimated, some closer judgment of this time lapse would be possible. Since we do not possess this knowledge, all that can be established is that the activating etiological factor must always be sought, not at the beginning of the deafness or at any period following its exacerbation, but months or years before the deafness occurred, increased, or became stabilized. It may be necessary to search for a factor preceding birth or in the family history. In my opinion the neglect of this clinical approach accounts in part for our abysmal ignorance of much of the etiology of otosclerosis and of its progression. We have failed because our laboratory and clinical studies have been focused too much on the present, when most or all of the signs and symptoms accompanying the incipiency of the lesion have faded. When suggestive symptoms and abnormal laboratory results have been found, I suspect that they were obtained during or near periods of sufficient activity of the factors involved to render them reflectable in any abnormalities observed. However, even in such instances, an exacerbation of the lesion may not have resulted in an increase in the deafness for some time. There are two lines of preclinical attack open to us: (1) family histories (heredity) and (2) personal histories, especially preclinical symptoms as revealed by emotional states, fluid and solid tissue chemistry, and neurocirculatory and metabolic variations. Clinical phenomena observed may be assigned etiological status when, in truth, they are merely coincidental or secondary in nature. Moreover, although they occur with onset, exacerbation, stabilization, recurrence, or cessation of other symptoms, their relationship to a disease may be one of effect rather than of affect (caused by rather than the cause of). Such may also be the case when they are related to preclinical observations.

CLINICAL FINDINGS

In otosclerosis the onset of deafness and its periods of rapid increase appear to coincide in time with, or more commonly subsequent to, puberty, childbearing, the onset of the menopause, severe illness, psychic traumas, and stress and anxiety, all of which are accompanied by major autonomic nervous system readjustments or dislocations. When a careful history is obtained, I have found that there is usually a considerable interval of time between these happenings and the deafness that follows them. I have never observed deafness from otosclerosis occurring after the menopause unless it had been present before then, but I have frequently observed an increase in the deafness following the menopause. The difficulty has been to relate properly such episodes to etiology, activation, and stabilization of the lesion and to evaluate their relative importance. In the pursuit of such evidence it should be kept in mind that, as in other investigations, any sustained and systematic effort to uncover data will probably be successful, at least in part.

During the past 12 years I have had under continuing observation seven pairs of identical twins, one or both of whom had otosclerosis in the right, left, or both ears. Recently two additional pairs of twins have been observed but as yet have not been sufficiently examined to be included in this discussion. Of these seven pairs of twins with otosclerotic deafness, I found that (1) in one pair (male), only the right ears were severely deafened; (2) in two pairs (female), all had deafness in both ears; (3) in two pairs (female), one twin in each pair had deafness in only one ear; and (4) in two pairs (female), one twin in each pair had no deafness in either ear, at my first examination. In two pairs of these identical twins no certain histories of differences in temperament or emotional sensitivity could be obtained. In both of these the otosclerotic deafness was first admitted at the same age, and both twins in both pairs had been subjected to difficult and frustrating conditions during their childhood and young womanhood for several years before deafness was noted. In five pairs there were definite differences

in the preclinical emotional histories of the twins of each pair. The twin who was the more sensitive or emotionally unstable before any deafness occurred was the one in whom deafness first developed or the only one of the pair in whom any deafness developed.

In one pair, the fourth above, who were 19 years old in 1946, one twin had high to average normal hearing in both ears even after the birth of two children (one in 1948 and the second in 1949). After the drafting of her husband and his assignment overseas, her left ear without any inflammatory episodes has during the past three years lost 30 to 40 decibels in the middle frequencies and 45 to 50 decibels in the lower frequencies. These losses still exist. There were marked emotional reactions for many months before I discovered this deafness. The hearing in the right ear remains high normal. Bone conduction remains normal in both ears. The second twin in her early teens was the more emotionally sensitive of the two. In 1946 she had average normal hearing for the middle and higher frequencies and low normal for the lower frequencies in the left ear. Her right ear showed a loss of 25 decibels at 4,000 cps but was within average normal limits for the middle speech frequencies and lower with each octave frequency for the lower tones, showing a loss at 125 cps of 30 decibels. There was a family history of progressive deafness. I made a diagnosis of otosclerosis. The second twin was married in 1952, and a child was born in March, 1953, without any further loss in her hearing in either ear to the seventh month of pregnancy, at which time I last examined her. She had received careful medical care during and subsequent to her pregnancy. In the other four pairs of identical twins, emotional stresses have been followed by varying differences between the twins in their right and left ears, but to avoid confusing the picture I shall omit such details and also those regarding fingerprints, audiograms, roentgenograms, otoscopy data, blood chemistry studies and various somatotype photographs, anthropometric data, blood types, basal metabolic rate, and data on sludging of the blood.

EXAMINATIONS

A word of caution in diagnosis: More than 200 twins were examined in the New York City public, primary, and high schools, and not a single case of deafness due to otosclerosis was detected, even though many of these children were subsequently reexamined by me. It is evident that such deafness in the child is rare indeed, and to diagnose it in children and even in young adolescents is usually impossible or very uncertain. How is it then that so many young patients are referred to me for an opinion on the propriety of having fenestration operations performed? Often parents tell me that the surgeon recommending fenestration has stated that their child has "fulminating otosclerosis," sometimes after but a single examination in cases in which I myself cannot make a diagnosis of otosclerosis but can account for the deafness by some other middle ear impedance lesion. A false or unwarrantedly pessimistic diagnosis may cause emotional repercussions in the patient and be more detrimental to a patient's health and hearing then the presence of the lesion itself.

SUMMARY AND CONCLUSIONS

In support of my belief that emotional factors are involved in the etiology of otosclerosis and in its exacerbation and extension, I submit at this time the following observations. Blood chemistry studies obtained during periods of stabilization seldom showed any impressive variations from the normal. However, a few very low values were observed, and two-thirds of my patients did show low normal values in either the serum calcium or the phosphorus levels. It is possible that these low values may have coincided with some changes in activity in the lesion.

Careful histories revealed that the incidence of otosclerosis was greater in the emotionally sensitive or unstable than in the more emotionally stable of the siblings in families with a history of otosclerosis. In large families I found the incidence of otosclerosis to be greater in the younger than in the middle or older brothers and sisters (especially so in females). In small families of two or three siblings the incidence was found to be greater in the older siblings. The older and the younger siblings are subjected to quite different problems, frustrations, and adjustments than the others. Many nontwins with otosclerosis revealed definite periods of excessive emotion before deafness, but it was difficult to determine the causative role, if any, of these emotional states. Nontwin and fraternal twin siblings differ from one another to a much greater degree, both in inheritance and in environment, than do identical twins.

As is generally known, puberty, childbearing, and the menopause frequently precede, by varying time intervals, the onset or the increase of a deafness caused by otosclerosis, and all are periods of major changes in body metabolism. Important chemical, endocrine, neurovascular, and psychological readjustments occur during these periods. Severe psychic shock from an accident, illness, loss of loved ones, divorce or quarrels of parents, and love affairs cause severer emotional stress and repercussions in the sensitive child than in the more emotionally stable child, adolescent, young adult, and even in the more mature person. Emotional instability or excessive stress can greatly affect both endocrine and chemical metabolism and sludging of the blood; easily detectable neurovascular phenomena such as an increase in the blueness of the sclera, flushing, and psychosomatic disorders commonly accompany these changes.

In the identical twins studied, in every instance but two, the twin in whom deafness due to otosclerosis first developed was the one who prior to the deafness was the more emotional of the two. In two instances otosclerosis developed in both twins at about the same time and both suffered severe emotional stress in their teens. In the otosclerotic patients, in addition to the histories of preclinical emotional instabilities, it was sometimes suspected that the emotional disorders were but one of the factors that might have been capable of precipitating the onset of the lesion or its progression. It is my opinion that emotional instability or endocrine or some other imbalance must be present before there is development or exacerbation of the disease.

140 E. 54th St. (22).

CECOSTOMY AND COLOSTOMY IN ACUTE COLON OBSTRUCTIONS

EXPERIENCES IN NINETY-NINE CASES

Frank J. Rack, M.D.
and
Kenneth W. Clement, M.D., Cleveland

The relative merits of cecostomy and colostomy as decompressive measures in acute large bowel obstruction have long been debated. During the past decade the management of large bowel lesions has undergone many changes; chemotherapeutic and antibiotic agents are now extensively employed, and single stage procedures with end-to-end anastomoses are considered in most instances the procedures of choice. However, when the lesion has produced complete obstruction, surgical decompression must still precede definitive surgery. Under these circumstances the surgeon is often confronted with the choice between two procedures, cecostomy or colostomy. For this reason it was considered worth while to review our experiences with these measures during the past 13 years at Cleveland City Hospital.

MATERIAL

Between Jan. 1, 1940, and Jan. 1, 1953, 99 patients with acute large bowel obstruction were admitted to City Hospital, Cleveland. During this period there were 161,765 admissions to the hospital and 27,241 admissions to the surgical service. City Hospital cares largely for the indigent residents of the city. The group under consideration not only is in the older age range but also represents a group that is often self-neglecting and is seen at the hospital when disease is further advanced than it is when seen in most private institutions. These patients were treated by different residents under the supervision of senior visiting surgeons. Although there was no routine established for the management of these patients, most patients underwent surgical decompression within 24 hours after admission. Cecostomy was carried out 54 times, colostomy 45 times. On one patient, both procedures were done: cecostomy originally, followed by resection, and colostomy two years later for an obstructive type of recurrence. The average age of the patients was approximately 65 years. The youngest patient was 44 and the oldest 90. Sixty-two per cent were men, and 38% were women. The anatomic distribution of the obstructing lesions in this series is listed in table 1.

All available roentgenograms were reviewed to determine the degree of small bowel distention associated with the acute colon obstruction. Thirty-five patients had no small bowel distention and were therefore considered to have a competent ileocecal valve. Thirty-four patients had a variable amount of small bowel distention ranging from minimal to maximal, suggesting an incompetent ileocecal valve. Vomiting recurring three or more times was recorded as a symptom in 56 patients and was noted as absent in 35 patients. Correlation was attempted between the presence or absence of vomiting and the status of the ileocecal valve as judged by the presence or absence of associated small bowel distention observed on the roentgenogram. Vomiting occurred in 51.4% of the patients with a competent ileocecal valve and in 82.3% of the patients with an incompetent valve. There were 18 (33.3%) deaths among the 54 patients treated with cecostomy and 11 (24.4%) deaths among the 45 patients treated with colostomy. The correlation between the type of anesthesia used and mortality is presented in table 2.

COMMENT

Two important objectives present themselves in the treatment of mechanical obstruction of the intestine: decompression for immediate treatment and elimination of the obstructing mechanism for definitive treatment. When the obstruction is complete, decompression must be given early consideration. To accomplish this most effectively, large bowel obstruction must be carefully distinguished from small bowel obstruction. In the latter, and in the absence of strangulation, "long tube" intubation or formal surgical exploration or both may be properly indicated. In acute large bowel obstruction neither of these measures has a place. It is impossible to adequately decompress large bowel obstruction with long tubes, and manipulation of the distended obstructed colon through a large abdominal incision is dangerous and often fatal. Reliance on such features as the simple scout film and the absence of vomiting to distinguish between these two types of obstruction may be misleading. In our experience, unless the lesion can be palpated in the rectum or seen through the sigmoidoscope, all patients with intestinal obstruction about whom there is a doubt as to the type or point of obstruction should be subjected to emergency barium studies of the rectum and colon. This will prevent dangerous errors in diagnosis and management.

Vomiting associated with intestinal obstruction has been described as a useful differential diagnostic point between large and small bowel obstruction. This was conceived on the basis that regurgitant vomiting develops early in small bowel obstruction and late in large bowel obstruction. Reflex vomiting, however, frequently occurs in large bowel obstruction, and it is difficult to distinguish this type from regurgitant vomiting. In correlating vomiting, competency of ileocecal valve, and mortality of surgical decompression, it is interesting to note that vomiting occurred in 51.4% of the patients with closed loop types of obstruction (probably reflex vomiting) and in 82.3% of the patients with an open ileocecal valve (regurgitant vomiting). However, of the 35 patients who showed no gas in the small intestine by scout film, 11 (31.4%) died

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after decompression, whereas 50% of the patients who showed either minimal or maximal amounts of gas in the small intestine died. These figures suggest that the presence of distention of the small intestine in association with colon obstruction makes the prognosis somewhat poorer and suggests a disease process of some duration. The surgeon is likely to be impressed with the need for early surgical decompression for those patients with colon obstruction with marked dilatation of the cecum, but patients with both large and small bowel distention also need early surgical decompression. The above figures again point out the fallacy of attempting decompression of acute colon obstruction with the long tubes.

Once the diagnosis has been established, and the point of obstruction is found to be proximal to the mid-transverse colon and the cecum is uninvolved, cecostomy is

TABLE 1.—*Distribution of Acutely Obstructive Lesions and Operative Mortality*

Anatomic Location	No. of Cases	Treated by Cecostomy		Treated by Colostomy	
		Survived	Died	Survived	Died
Hepatic flexure	2	1	1	0	0
Transverse colon	2	2	0	0	0
Splenic flexure	7	5	0	2	0
Ascending colon	1	1	0	0	0
Descending colon	8	5	2	1	0
Sigmoid	42	11	12	16	3
Rectum	25	8	2	10	5
Site unknown	12	3	1	5	3
Totals	99	36 (36.3%)	18 (18.2%)	34 (34.3%)	11 (11.2%)

TABLE 2.—*Type of Anesthesia and Mortality from Cecostomy and Colostomy*

Anesthesia	Cecostomy		Colostomy	
	Living	Dead	Living	Dead
Gas, oxygen, and ether	5	0	2	1
Local infiltration	14	9	11	5
Continuous spinal	1	0	2	1
Continuous spinal and gas, oxygen, and ether	0	1	0	0
Spinal	15	5	15	3
Intercostal block	0	0	1	0
Pentothal and local	1	1	3	1
Local and gas, oxygen, and ether	0	2	0	0
Totals	36	18	34	11

the logical operation. If the cecum itself becomes obstructed, we believe emergency end-to-end anastomosis of mid-ileum to divided transverse colon is the procedure of choice. The distal ileal segment and the proximal portion of the divided transverse colon can then be brought out through stab wounds and right colectomy completed at another operation. If the patient's condition is good, the entire procedure, including resection of the right colon, can be completed as an emergency. If the point of obstruction is found to be in the left transverse colon, splenic flexure or proximal descending colon transverse colostomy may be done as well as cecostomy, but satisfactory resection of lesions in these sites, along with their lymphatic drainage areas, is very difficult, if possible at all, in the presence of a transverse colostomy. Cecostomy is preferable for obstructing lesions in these areas.

Obstructing lesions in the descending colon, sigmoid, rectosigmoid, and rectum may be decompressed in several ways. If the lesion can be palpated in the rectum or visualized in the sigmoidoscope, a simple expedient is to attempt to pass a heavy catheter past the point of obstruction through the sigmoidoscope. This must be done carefully, lest a perforation be made, but, if the attempt is successful, it allows for decompression and preparation of the patient for definitive surgery without preliminary decompression. If the lesion is impassable or if it lies above the range of the sigmoidoscope either cecostomy or colostomy may be done. Comparative mortality figures for these two procedures reported to date are inconclusive (table 3). Our own series reveals little significant difference in mortality for the two procedures. While making a cecostomy, there is commonly a variable degree of fecal contamination, whereas a colostomy can be performed with comparatively little spill either immediately or several hours after exteriorization. In our series there were seven proved cases of death due to peritonitis following surgical decompression: four after cecostomy and three after colostomy.

TABLE 3.—*Mortality Following Colostomy and Cecostomy*

Authors	Number of Cases		Mortality	
	Cecostomy	Colostomy	Cecostomy	Colostomy
Rankin [2]	16	..	18.7%	.....
Gregg and Dixon [3]	31	35	9.7%	14.3%
Wangensteen [4]	..	..	50.0%	.....
Collier and Ransom [5]	7	..	71.4%	.....
Hendricks and Griffin [6]	142	47	40.5%	21.0%
Michel and McCafferty [7]	87	13	30.0%	15.3%
Albers and Smith [8]	34	34	56.0%	24.0%
Rack and Clement	54	45	33.3%	24.4%

It has been our experience that a cecostomy rather quickly decompresses the colon, but repeated irrigations over several days are required for the colon to function independently and to return feces. The most effective cecostomy in the series of patients reported here is the type done through a McBurney incision in which a Pezzer catheter is placed in the cecum through a stab wound. The colostomies in this series were either of the simple loop variety or of the Wangensteen type.[1]

SUMMARY AND CONCLUSIONS

Cecostomy and colostomy were done 99 times for acute colon obstructions in this series, with a mortality rate of 33.6% for cecostomy and 24.4% for transverse colostomy. Lesions proximal to and in the splenic flexure that produce acute obstruction are best treated with emergency cecostomy. Transverse colostomy in the pres-

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ence of lesions in the splenic flexure makes satisfactory definitive surgery difficult. For obstructing lesions distal to the splenic flexure, adequate decompression may be effective by either cecostomy or transverse colostomy; however, cecostomy has a somewhat higher immediate mortality rate than transverse colostomy. In general, we prefer cecostomy for obstructing lesions of the left colon for the following reasons: 1. With cecostomy the area of bowel most susceptible to perforation is decompressed earliest. 2. The mild bacterial contamination of the peritoneal cavity incident to cecostomy provides immunity for future surgery. 3. Bowel preparation and depression of bacterial flora can be satisfactorily done with a cecostomy. 4. In most instances after definitive surgery the cecostomy usually closes spontaneously and does not require a major surgical procedure for repair as does colostomy. The mortality incident to closure of a temporary colostomy must be considered in evaluating comparative mortality figures for cecostomy and colostomy.

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INTRAPARTUM INITIATION OF LACTATION CONTROL WITH A LONG-ACTING ANDROGEN

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The mammary glands are indeed an integral unit of the female reproductive and sexual system. Were it not for the evolution and development of the function of these structures, it is conceivable that some form of life other than man would dominate this planet. In the female of our species the breasts are erotic centers, and, under certain circumstances, their stimulation may arouse different degrees of sexual excitement as well as induce contractions of the uterine musculature. Most important of all, however, they are the organs that assume the nourishment of the offspring after it emerges from its intrauterine habitat where it was maintained and nurtured through the maternal and placental vascular arrangement. Soon after nidation of the fertilized ovum, preparations are begun so that the breasts may take on their principal function some months hence when the mechanism of lactation is initiated almost simultaneously with the cessation of the mechanism of labor. Thus is provided a continuous means of sustaining each successive generation. The increased estrogens produced by the corpus luteum of pregnancy stimulate the production of a lactiferous duct growth factor or mammotropin of pituitary origin. An alveolar growth mammotropin is also formed from the same source through the combined stimulation of estrogens and progestin.[1] From an early stage of pregnancy, then, the ovarian hormones in cooperation with the pituitary gland bring about full development and priming of the breasts during the first 120 days. Thereafter they remain in relative status quo until the termination of pregnancy.

The activation of lactation occurs when the anterior pituitary lactogenic hormone, called galactin or prolactin, is released after restraint by the ovarian and placental estrogens throughout pregnancy. Upon the termination of the third stage of labor and the consequent rapid decline of the estrogen blood level during the first eight postpartum hours,[2] prolactin is increased and liberated, and within three to five days milk is secreted by the breasts. This interrelationship of the hypophysial gland and lactation was first observed in 1921 by Evans and Long.[3] Subsequently Selye, Collip, and Thomson,[4] Gomez and Turner,[5] and Riddle [6] demonstrated the control of lactation by the pituitary gland. The nervous stimulation of sucking and the removal of the milk are also essential to its sustained production, and, if the breast is not suckled postpartum, suppression of lactation eventually occurs in spite of the copious fount of lactogenic hormone. Extraordinary instances of the establishment of lactation in nonpregnant women simply by sucking of the nipples have been quoted by Greenblatt.[7]

The decision that a newborn infant is not to suckle but is to receive artificial nursing instead is just as often dictated by the will of the mother as it is by the judgment of the attending physician. Some women are decidedly unwilling to suckle their infants for reasons that may be imperspicuous or, on the other hand, for reasons that are understandable and apparent. It is our opinion that mothers who refuse to nurse their babies should not be urged to change their attitudes except for some extraordinary reason. However, there are cogent reasons why the newborn baby may not be put to his mother's breasts. Such disorders as deformed or chronically sore or fissured nipples, or abscess during a previous pregnancy, preclude nursing. Some maternal systemic and weakening diseases such as tuberculosis, myasthenia gravis, a malignant condition, or certain anemias and psychoses may contraindicate nursing. There are also instances when nursing would be most welcomed and natural, but where lactation must be suppressed because of the prenatal or perinatal loss of the baby.

Lactation usually produces engorgement of the breasts on the third, fourth, or fifth day of the puerperium, and, when the full mammary glands are not relieved by emptying, they become tense and swollen, heavy and tender. The painful turgescence persists from 48 to 72

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Testosterone cyclopentylpropionate in cotton seed oil was made available for this clinical study and evaluation by Dr. H. F. Hailman of the Upjohn Company, Kalamazoo, Mich.

hours. Usually it is the cause of considerable discomfort and distress to the patient, and, if proper attention is not given, acute inflammation and subsequent abscess formation may ensue.[8] As early as 1926 Parkes and Bellerby [9] demonstrated the inhibitor effect of estrogen on the established lactation of mice. Since that time, many other investigators [10] have confirmed these observations and have made similar deductions from their work with other animals. Before 1938, when postpartum lactation was not desired, the patient was restricted in her fluid consumption, and the local application of ice, saline purgatives, intramuscular injections of camphor in oil, and the administration of analgesics were often used to provide a measure of relief.

In 1938 Foss and Phillips [11] suggested the use of estrogen to inhibit lactation in women, and it has been widely adopted in various forms and doses during the last 15 years.[12] Repeated administration of the hormone is required during the puerperium, but its discontinuance within a few days usually causes the appearance of congestion of the breasts, thus merely delaying the symptoms rather than avoiding or minimizing them. Also, discontinuance of the estrogen therapy is usually followed by excessive uterine (withdrawal) bleeding, and nausea may be an unpleasant side-effect. In a recent clinical experiment by Kosar, Nash, and Buxton,[13] dienestrol was found to have had a lesser inhibiting effect on lactation than simple symptomatic therapy. The androgens like estrogen will also suppress lactation. In 1937 Robson [14] was able to stop lactation in the nursing mouse by the injection of 0.1 mg. of testosterone propionate. The next year Kurzrock and O'Connell [15] used the same preparation to arrest lactation in some of their patients. They used from 50 to 100 mg. in divided doses of 10 to 25 mg. each, beginning on the third postpartum day. We and others [16] have also successfully used divided doses of testosterone propionate given parenterally, and androgen-estrogen combinations have been used parenterally by Katzman [17] to prevent pain and lactation during the puerperium. All of these methods, however, require repeated injections.

More recently, and until October, 1952, we have used methyltestosterone, prepared for buccal absorption, to suppress lactation. Our patients were given five 10 mg. tablets on the second postpartum day, and they were asked to place one under the tongue every two hours until all five had been used. The procedure was repeated the next day. Some patients found them objectionable, but the results by this method were satisfactory. The congestion of the breasts lasted from 24 to 48 hours, but occasionally longer. The mastalgia was reduced to a tolerable degree. The wearing of a breastbinder, the application of ice, and the oral administration of codeine sulfate combined with acetylsalicylic acid gave additional relief. There were no instances of subsequent filling of the breasts after they had become decongested, and there was no withdrawal bleeding. The administration of methyltestosterone sublingually was superior to the oral administration of diethylstilbestrol for the purpose for which it was employed, but repeated and frequent administrations were also necessary.

In October, 1952, we decided that it might be possible to initiate the control of lactation before delivery, since, at the time of admission to the hospital of certain patients who were in labor, we or they knew that they were not going to nurse their babies. Thus could be bridged the important time lag between the termination of pregnancy with its consequent sudden and rapid removal of the chorionic source of the lactation inhibitor, and the usual 48th postpartum hour, when the first dose of the inhibiting medication is usually administered. During this important period the lactogenic hormone is being rapidly increased in the anterior pituitary gland and liberated. It appeared to us, too, that an injection during labor of an optimum dose of a long-acting androgen such as testosterone cyclopentylpropionate in cottonseed oil might produce and maintain a lactogenic-inhibitory-hormone level in the blood stream, which would obviate the necessity of repeated administrations and which might appear in the blood stream before the liberation of lactogen. The cyclopentylpropionic acid ester of testosterone was found by Ott [18] and his associates to have had a period and degree of effectiveness that was markedly in excess of that of testosterone propionate. Lloyd and Fredericks [19] reasoned from their studies with the same androgen in oil that the lesser effectiveness of testosterone propionate was probably due to its production of such a rapid rise and fall in the circulating steriod level that it could not be efficiently utilized by the patient. The ester of testosterone cyclopentylpropionate is inactivated at a slower rate, which permits more economic utilization, and the steady effective level so produced is maintained for a greater period.[20]

There were 125 patients in this study, 49 of whom were private patients of two of us (S. M. D. and J. M. F.); and, of the rest, some were staff patients but the majority were private patients of other members [21] of the attending and courtesy staffs of the department of obstetrics and gynecology of George Washington University Hospital, who cooperated in this study and gave us the benefit of their own observations and impressions and their patients' reactions. Nearly all of the patients included in this experiment had answered "no" on their admission in labor, when interrogated as to whether they expected to nurse their babies, or it was known that there would be no need to allow lactation. One hundred of them were given 100 mg. of testosterone cyclopentylpropionate (Depo-testosterone) intramuscularly when labor was definitely established. This usually corresponded with the time that the first dose of obstetric analgesia was administered, and averaged five hours before delivery. Twenty-five of the patients received the preparation after delivery, at some time during the first eight postpartum hours as an average.

Daily recorded observations were made on each patient by two of us (P. A. S. and H. L. M.). Notes were kept concerning the degree and duration of breast engorgement, if any, no matter how slight, and of the maximum number of hours of real discomfort that each patient experienced while she was in the hospital. Subsequent inquiries were made of the attending physicians concerning any breast complications that may have arisen while their patients were convalescing in their own homes. We were also interested in knowing what each patient required to support her breasts—her own brassière or a hospital breastbinder. Any patient who

had had a previous experience with the suppression of lactation was asked to compare that experience with her present one, and her comments were summarized. We consider the number of hours of pain or discomfort from fulness of the breasts as a satisfactory index of the value of our therapy, since uninhibited lactation will usually cause the breasts to be painful and turgid for several days unless the milk is removed or until failure to empty them produces stasis and ultimate involution. In cases in which the discomfort persisted for longer than 24 hours the result was not to be considered satisfactory.

One hundred thirteen, or 90.4%, of the 125 patients in this group did not complain of any discomfort in their breasts for more than 24 hours, although swelling occurred in all except 5 cases. Forty-one did not complain at all, and 72 reported discomfort for less than 24 hours. Only 12 patients could be classified as not having been helped, since they were uncomfortable for more than 24 hours. One hundred eleven patients were able to wear their own brassières during the entire postpartum period, which in itself is an additional indication of the minimal swelling permitted by the action of the preparation that we studied. Hospital breastbinders were required for the other 14 patients. No limit was placed on either the amount or the nature of the patients' fluid intake.

There were no incidents of delayed breast congestion in any of the entire group after the initial engorgement and stasis had subsided, and there were no cases of mastitis. Each of six patients, after returning to her home, reported that there had been one day when milk was secreted by her breasts, but in each case it was unassociated with any swelling or discomfort, and no treatment or special care was required. One patient reported the appearance of milk for six weeks. Six other patients lactated in the hospital for periods of less than 48 hours. In one patient a noninflammatory lactocele developed that was somewhat painful but that absorbed spontaneously. From the 42 women who were able to compare the effects of testosterone cyclopentylpropionate with other methods of lactation control, the favorable opinion of 36 was "better" or "much better," while the remaining 6 felt there was little difference.

We observed that there was a decided absence of the uncontrollable passing depression that some women experience sometime during the first week of the lying-in period and that has been referred to by the colloquialism, "fifth day blues." The favorable protein anabolic action attributed to androgens was probably responsible. The single 100 mg. dose of androgenic hormone such as we used was too small to have any masculinizing effects on the adults of this series, and the preparation that we used did not cause any local irritation or unpleasant systemic reactions.

SUMMARY AND CONCLUSIONS

Our experiment reveals that a single injection of 100 mg. of testosterone cyclopentylpropionate in cottonseed oil (Depo-testosterone) given intramuscularly during the intrapartum or early postpartum period is an altogether sufficient and satisfactory method to adopt for the control of lactation and to minimize the degree and duration of the pain caused by puerperal breast engorgement in nonnursing mothers. The results are better than heretofore obtained by the repeated administrations of estrogen and other forms of androgen, and the single dose method is more acceptable to the patients.

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BODY TEMPERATURE STUDIES IN ANESTHETIZED MAN

EFFECT OF ENVIRONMENTAL TEMPERATURE, HUMIDITY, AND ANESTHESIA SYSTEM

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The possibility of great harm occurring as a sequel to marked elevations of body temperature is well known. Heat retention (also called heat stroke or heat pyrexia) may be associated with convulsions, coma, and psychic disturbances in the recovery phase.[1] In fatal cases, generalized degeneration of the neurons of the central nervous system, centrolobular liver necrosis, lower nephron nephrosis, and widespread hemorrhages, especially in the lungs and subendocardial tissues, are found.[2] The similarity of the pathological lesions found in patients dying following fever therapy and those produced by anoxia from any cause has been stressed by Hartman [3]; hypoxia occurs at the tissue level because of increased metabolic oxygen demand associated with fever, which cannot be met by the cardiorespiratory transport system.

It has been suggested that liver cells in addition to the cells of the central nervous system are particularly susceptible to damage during high fever.[4] Jaundice is a well-recognized complication of fever therapy, and several deaths have occurred following such treatment in which extensive liver necrosis was found at autopsy. Lesser elevations of fever produce apparently reversible impairment of liver function.

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In addition, hyperthermia imposes a severe stress on the cardiovascular system. Heating the entire skin area may lower the over-all peripheral resistance to two-thirds or half of normal, according to Bazett.[5] In order for an effective general circulation to be maintained, the cardiac output increases and splanchnic vasoconstriction occurs.[6] These cardiovascular responses to heat decrease the ability of the body to withstand further circulatory stress. Experimentally, exposure to a hot environment sufficient to cause a rise in body temperature decreases the ability of animals to survive hemorrhagic shock,[7] traumatic shock,[8] and shock secondary to thermal burns.[9] External thermal stress adversely affects the circulatory status of patients in congestive heart failure, as has been shown by Berenson and Burch; in normal subjects circulatory responses to positional change are impaired.

This clinic has been particularly aware of the serious nature of the problem of heat disorders during anesthesia for many years because of high temperatures and humidity in the operating rooms during several months of the year. Despite vigilance, in the past two years alone two children and one adult have had convulsions associated with hyperpyrexia while anesthetized. Certain similarities were notable in all of these cases. All occurred during the summer months, with the temperature of the operating room near 90 F and the relative humidity high. All three patients were acutely ill, having had peritonitis for 36 hours or longer before surgical intervention. The cause of the peritonitis was a perforated appendix in two cases, and was due to leakage from the duodenal stump following subtotal gastrectomy in the third. In every case, convulsions were preceded by a rapid, progressive rise in body temperature. Fortunately, prompt treatment directed toward controlling the convulsions, rapidly reducing the fever, and providing maximal oxygenation averted a fatal outcome in each instance.

TABLE 1.—*Age and Sex Distribution, Anesthetic Agents Used, and Duration of Anesthesia in Heat Study Cases*

	Type of Anesthesia System			
	To-and-Fro	Circle	Non-Rebreathing	Total
No. of Cases	67	54	43	164
Ratio of Males:Females	39:28	32:22	29:14	100:64
Age, Yr.				
12-20	4	..	7	11
21-40	10	13	9	32
41-60	26	20	9	55
61-70	21	14	11	46
Over 70	6	7	7	20
Anesthetic Agent				
Cyclopropane	58	41	..	..
Ether	9	13	..	..
Nitrous oxide-oxygen-thiopental	..	..	39	..
Nitrous oxide-oxygen-trichlorethylene	..	..	3	..
Nitrous oxide-oxygen-ether	..	..	1	..
Duration of Anesthesia				
Mean	3 hr. 57 min.	4 hr. 8 min.	4 hr. 34 min.	4 hr. 13 min.

The dangers of hyperthemia following anesthesia in children have recently been emphasized by Bigler and McQuiston.[10] Two deaths in infants attributable to this cause were reported. Knight [11] observed rises as high as 5 to 7 degrees F in body temperature following operations performed in the summer. These temperature rises appeared to be associated with elevated room temperature, prolonged surgery, heavy draping, and the use of the carbon dioxide absorption system. Burstein and Mark,[12] as well as Knight, have advocated cooling the inspired gases when to-and-fro or circle absorption systems are used in hot weather. The observations of rectal temperature during anesthesia made by Searles and Lenahan [13] show in a general way the effects of environmental conditions on body temperature.

No systematic study has been made to show the behavior of body temperature in the anesthetized subject under various conditions of room temperature and humidity. It would be desirable to know the critical temperature and humidity conditions above which heat retention becomes a frequent complication. Moreover, no clear relationship has been demonstrated between body temperature changes and choice of anesthesia system. The present study was undertaken to provide more definite information about these matters that might be of value to the anesthesiologist and surgeon.

CLINICAL MATERIAL AND METHODS

Temperature measurements were made in 212 general hospital patients undergoing surgery and general anesthesia. The records of 164 of these patients were selected for a study relating body temperature changes with environmental temperature, humidity, and choice of anesthesia system. The criteria used in selecting these cases were as follows: Only cases in which surgical procedures lasted more than two hours were used. The subjects were patients weighing more than 90 lb. (40.9 kg.) who had no fever before operation and who showed no evidence of shock during the procedure.

The duration of anesthesia varied from two to nine and one-half hours; the average for all cases was slightly over four hours. The age of the subjects ranged from 13 to 85 years. Age and sex distribution are presented in table 1.

Small children were excluded because their smaller body mass and different physiological responses to heat render them unsuitable for comparison with adult subjects. Patients with fever were excluded from the study because it was assumed that they might differ from afebrile subjects in their responses to thermal stress. Rectal temperature was used as a measure of internal body temperature in 150 cases; gastric temperature was used in 9 cases; and low esophageal temperature was used in 5 cases. The mean body temperature for all sub-

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jects at the beginning of anesthesia was 99.2 F. The variation was from 97.0 to 100.8 F; however, less than 4% of cases fell outside of the range 98.0 to 100.2 F.

Patients in whom signs of hemorrhagic or traumatic shock developed during the course of the operation were not included in the study. Body temperature was regularly observed to fall after the onset of shock in these cases, and their inclusion was not considered permissible.

All patients except six received preanesthetic medication within 90 minutes of induction of anesthesia. Of these, all received a therapeutic dose of a belladonna drug, which in over 93% of cases consisted of 0.4 mg. of atropine. Meperidine was given to 52% of the patients in addition, and 35% received morphine.

Inhalation anesthesia, administered by endotracheal technique, was used in all cases. Three different anesthesia systems were used: to-and-fro absorption, circle absorption, and non-rebreathing technique. The first two mentioned were employed in a closed system in all cases in this series. The Stephen-Slater valve,[14] which permits no rebreathing of exhaled gases, was used in the third group.

The anesthetic agent employed with the to-and-fro and circle systems was either cyclopropane or diethyl ether. Cyclopropane was used approximately four times more frequently than ether, with both the to-and-fro and the circle systems. More than 90% of the patients anesthetized with the non-rebreathing technique received nitrous oxide and oxygen supplemented with sodium thiopental. In the remaining cases either trichlorethylene or ether was the supplement (table 1). Flows of 6 to 12 liters per minute of nitrous oxide-oxygen were required; concentrations of nitrous oxide above 75% were not employed.

This study was conducted during the course of one calendar year. Thus it was possible to assess the responses of anesthetized patients to a graded series of thermal stresses related to seasonal changes. The operating suite in which the study was made is not air-conditioned. Instruments are sterilized by boiling in a room that opens directly upon two adjacent operating rooms. This arrangement results in considerable humidification of the room air. The overhead operating lights in use utilize four 250 watt incandescent lamps. The sterile draping system in practice in this hospital was used consistently throughout the study: four small doubled sheets ("clinics") were placed on the four sides of the field; one sheet, folded in half, was then placed respectively across the foot and one across the head of the table; finally, a laparotomy sheet was placed on top of these drapes.

The wet and dry bulb temperature of the room air was measured to within 0.5 degrees F with a sling psychrometer. Measurements were made at approximately 90 minute intervals during the procedure. The relative humidity was computed from psychrometric tables.

Body temperature, and temperature within the anesthesia system and the airway, was measured with a Micromax 6 point recording thermocouple (manufactured by the Leeds and Northrup Co., Philadelphia). Copper and constantan wire thermocouples were used. The accuracy of this machine is ± 0.3 degrees F. A United States Bureau of Standards calibrated mercury thermometer was used to check the performance of the machine, and during many of the measurements, one thermocouple was kept in an ice bath as a reference point. The latter was desirable, although not essential, inasmuch as the machine utilizes a self-contained, known electromotive force instead of a cold junction as a basis for comparison with the electromotive force produced by the test thermocouple.

Temperature was recorded between points at one minute intervals; hence, the time between successive readings on any one thermocouple was six minutes. The rectal thermocouple, mounted in a plastic rod, was inserted 4 or more inches into the rectum at the earliest possible moment after anesthesia was established. Gastric or esophageal temperatures were obtained with a thermocouple mounted in a length of vinyl plastic tubing (2.5 mm. external diameter), which had been stiffened by treatment with heat. Fine, unsheathed, insulated thermocouple wires were used to obtain temperatures in the airway and in anesthesia equipment. Low tracheal or carinal temperature was obtained by inserting such a wire a measured distance beyond the tip of the endotracheal airway, usually 3 to 4 cm. Temperature in the to-and-fro cannister was measured in the proximal one-third of the cannister. Measurements were made at several locations in the circle cannister, through holes drilled in the side; only measurements made in the proximal one-third are described herein. Temperatures were measured in the accordion tubes of the circle machine. Two thermocouples were placed in the inspiratory tube, one 4 in. from the Y piece and the other adjacent to the machine; two other thermocouples were similarly placed in the expiratory tube. The accordion tubes used were 31 in. (77.5 cm.) long. The temperature under the drapes adjoining the chest, the flank, the hip, and between the thighs was measured. Thermocouples were placed about 3 cm. from the body and were protected from moisture.

The mean temperature was computed from a series of observations in a number of patients, six minutes apart over a period of two or more hours. Two to-and-fro, or two circle cannisters were alternated at hourly intervals, and were not cooled. Data upon which mean cannister temperature was based were only those observed when the patient was breathing through the test cannister, not during the cooling period of the cannister.

RESULTS

The effects of room temperature, humidity, and anesthesia system on body temperature were analyzed in the following way. Body temperature change, whether a rise or a fall from the initial recorded internal temperature, was plotted against the duration of anesthesia in hours. The body temperature records were then grouped in two general ways, according to environmental conditions: first, by the dry-bulb temperature of the operating room (in 5 degree F ranges), and each range divided in two subgroups according to whether the relative humidity was above or below an arbitrary figure of 55%; second, solely by the wet-bulb temperature of the room. The distribution of patients by these two methods is shown in tables 2 and 3 respectively.

14. Stephen, C. R., and Slater, H. M.: Nonresisting Nonrebreathing Valve, Anesthesiology 9: 550, 1948.

Figures 1 and 2 show the manner in which the data for each temperature range were plotted; some 30 other charts cannot be shown because of limitations of space. Each dot on the scatter chart represents the body temperature of one patient at the time shown; the average temperature change is shown by a curve. Temperature

TABLE 2.—*Distribution of Heat Study Cases According to Dry-Bulb Temperature Range (F), Relative Humidity, and Anesthesia System*

	Type of Anesthesia System			
	To-and-Fro	Circle	Non-Rebreathing	
Relative Humidity Below 55%				
75-79.5	6	12	4	
80-84.5	18	21	17	
85-89.5	12	7	8	
90-95	7	3	3	
Relative Humidity Above 55%				
80-84.5	5	5	5	
85-89.5	16	6	3	
90-95	3	..	3	
Totals	67	54	43	Grand Total 164

TABLE 3.—*Distribution of Heat Study Cases According to Wet-Bulb Temperature Range and Anesthesia System*

	Type of Anesthesia System			
	To-and-Fro	Circle	Non-Rebreathing	
Wet-Bulb Temperature, F				
58-64.5	8	17	11	
65-69.5	15	19	11	
70-74.5	19	8	11	
75-82	25	10	10	
Total	67	54	43	Grand Total 164

measurements were actually made at 6 minute intervals; however, in the interests of clarity, the interval shown on the charts is 18 minutes.

One fact immediately apparent is the wide variability of rate and degree of body temperature rise shown by different patients. The slope of the temperature curve for any particular patient is apt to be bumpy and irregular,

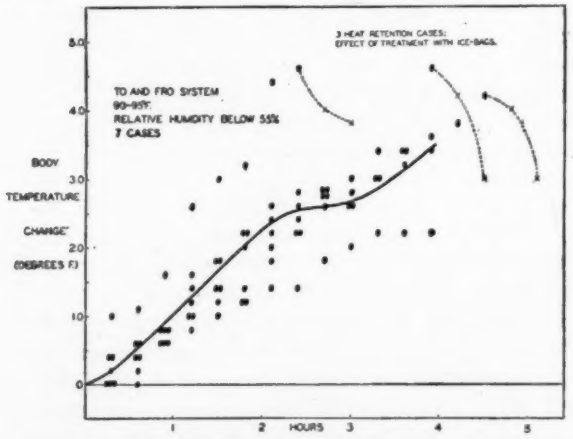

Fig. 1.—Body temperature changes during anesthesia in seven cases with the temperature of the operating room ranging from 90 to 95 F.

apparently related to the variable degree and intermittent character of sweating seen in these subjects. A definite trend in the course of the body temperature is usually discernible however.

In figure 1, at a dry-bulb temperature of 90 to 95 F with the relative humidity below 55%, three out of seven patients anesthetized with the to-and-fro system had heat retention requiring therapy with ice-bags. This occurred after 2.5 hours, 4 hours, and 4.5 hours of anesthesia. Significantly, the average wet-bulb temperature for the seven cases was 76.5 F. Only part of the cooling curve

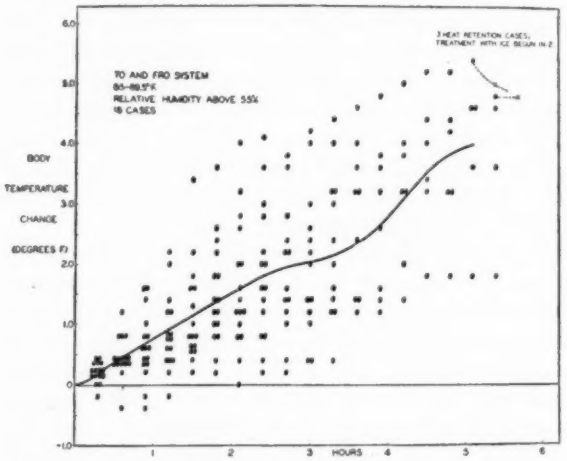

Fig. 2.—Body temperature changes during anesthesia in 16 cases with the temperature of the operating room ranging from 85 to 89.5 F.

of the treated patients is shown; however, the effectiveness of therapy can be clearly seen.

At 80 to 84.5 F, with the relative humidity above 55%, two of five patients anesthetized with the to-and-fro system were on the verge of heat retention after 3.5

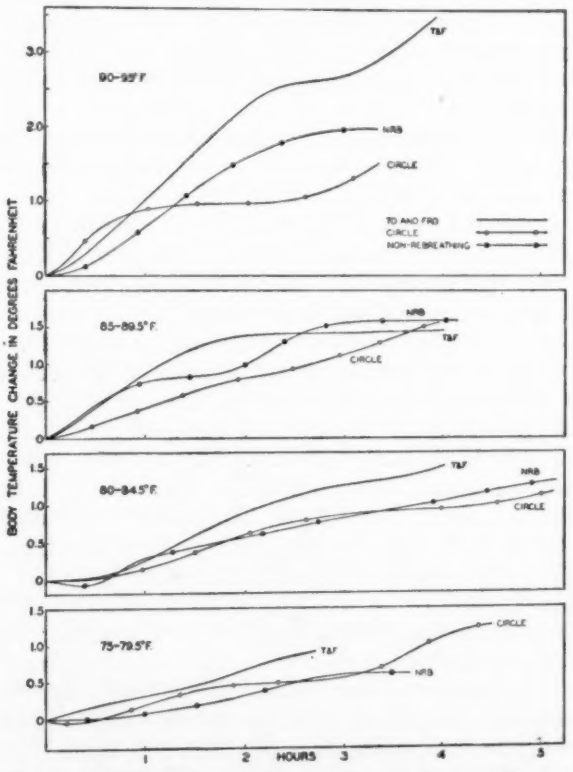

Fig. 3.—Average body temperature curves during anesthesia with the operating room temperature at various ranges but the relative humidity always below 55%.

hours (not shown). The average wet-bulb temperature was 74 F. Heat retention was averted in one patient by the application of ice-bags, and in the other when severe hemorrhagic shock supervened.

Heat retention occurred in 3 out of 16 patients in whom to-and-fro absorption was used at 85 to 89.5 F, with the relative humidity above 55% (fig. 2). The average body temperature rise for the group as a whole was over 4 degrees F in five hours. The average wet-bulb temperature was 77.5 F.

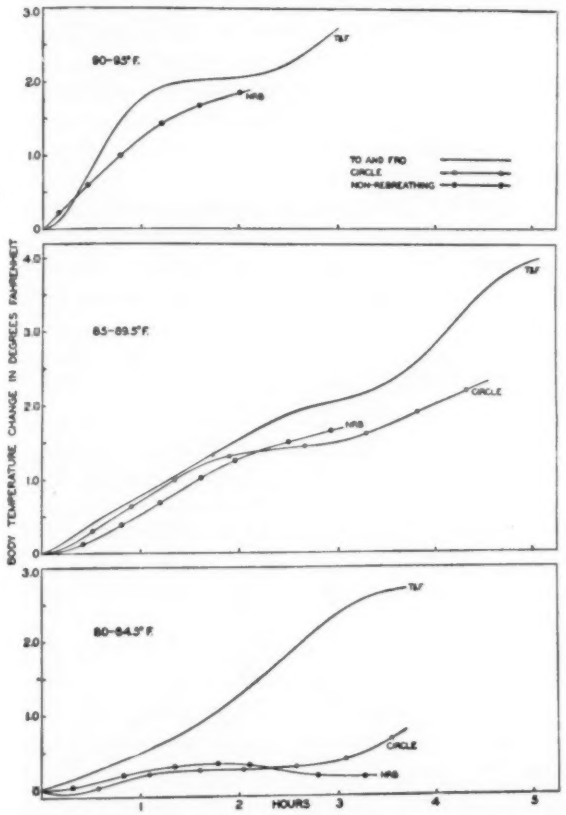

Fig. 4.—Average body temperature curves during anesthesia with the operating room temperature at various ranges but the relative humidity always above 55%.

At 90 to 95 F with the relative humidity above 55%, the body temperature rose at a rate of over 1 degree F per hour in three patients anesthetized with the to-and-fro system (not shown). The average wet-bulb temperature was 81 F. Heat retention developed in only one patient, but in both of the other two it was imminent at the time surgery was terminated. Under comparable conditions, heat retention developed in only one patient anesthetized with the circle or non-rebreathing system.

The average body temperature curves for the data grouped by dry-bulb temperature range, with the relative humidity below and above 55%, are summarized in figures 3 and 4.

Average body temperature curves plotted solely in relation to the wet-bulb temperature are shown in figure 5. From the statistical standpoint and for reasons that will be discussed, this figure is the one best suited for comparing the heating effects of the three anesthesia systems.

At wet-bulb temperatures below 75 F, the height and rate of body temperature rise were slightly but consistently greater in patients anesthetized with the to-and-fro system than when the circle and non-rebreathing systems were used. Above 75 F, this effect was considerably more marked. The rate and height of body temperature rise was similar with both the circle and non-rebreathing systems, although it was sometimes slightly higher with the latter.

The results of air temperature measurements under the drapes, adjoining the subject's body, are shown in table 4. The temperature between the interradiating surfaces of the thighs approached body temperature closely; only measurements made adjacent to the chest, flank, and hip are included in the results shown in the table. The air temperature under the drapes was 8 to 10 degrees warmer than room air when the latter was 80 to 90 F. Above a room temperature of 90 F, the temperature under the drapes approached body temperatures. The results of temperature measurements made in the airway, and in various locations in the three anesthesia systems studied, are shown in tables 5 and 6. Attention is called to the variation in temperature observed. The manner in which average temperature was computed has been described. The average temperature relationships within the three systems are summarized in figure 6.

TABLE 4.—*Average Air Temperature Observed Under Surgical Drapes*

Room Temperature, F	Under Drapes, F	No. of Patients	No. of Measurements
75-79.5	89	22	586
80-84.5	93	30	1,194
85-89.5	90	11	419
90-95	98	13	809

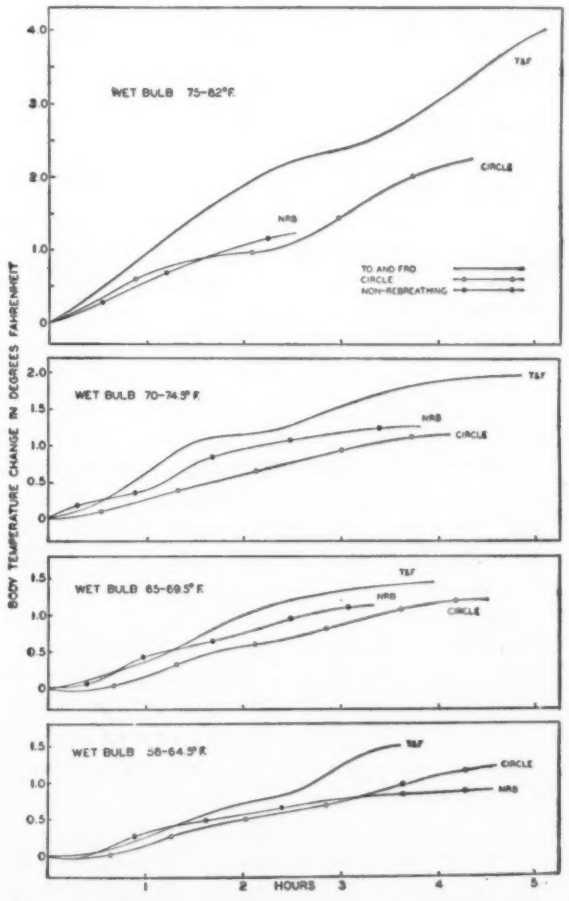

Fig. 5.—Body temperature changes with different anesthesia systems at various wet-bulb temperatures of the operating room.

COMMENT

Some of the undesirable effects that may result from heat retention have been mentioned. This type of heat disorder arises from a disturbance of the normal balance between heat gain to the body and heat loss.

The principal source of heat gain is internal heat production from the combustion of carbohydrate, protein, and fat, and to a minor degree from the specific dynamic action of protein. In addition, fever per se increases metabolic heat production by about 7% per degree Fahrenheit. Surgical patients may also gain heat from sources outside the body by radiation and conduction. Examples are the radiant heating effects of the operating lights and the warm bodies of the surgeons on the one hand, and heat gain from warm objects such as repeatedly applied hot laparotomy pads on the other.

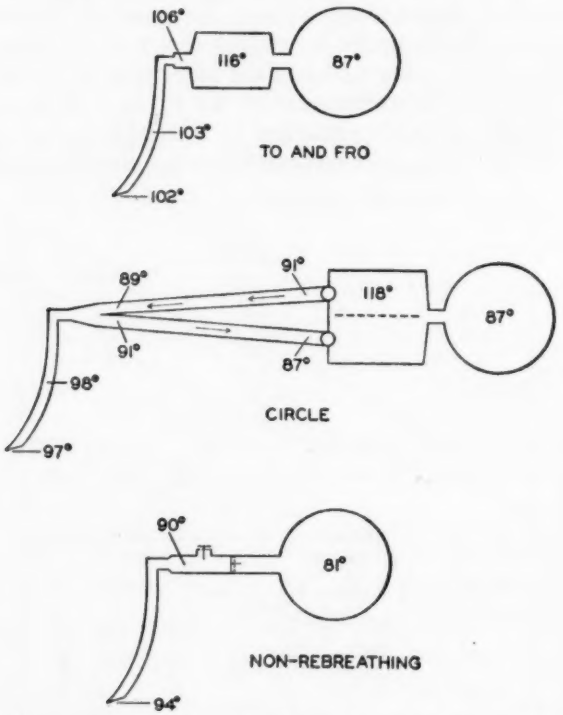

Fig. 6.—Average temperature (Fahrenheit) at various points in the to-and-fro, circle, and non-rebreathing anesthesia systems.

For thermal equilibrium to exist, the summation of factors resulting in heat gain must be balanced by an equal loss of heat by the body. The significance of a rising body temperature during anesthesia is a disparity between these two factors. Heat loss is mediated solely by four physicial processes: radiation, convection, conduction, and evaporation.

Heat loss by radiation is influenced chiefly by two factors: first, the difference in surface temperature of the subject and the objects surrounding him and, second, the effective radiating surface area. This area is always less than the total body area because of interradiating effects. Radiation is the principal means of body heat loss at comfortable room temperatures. However, its importance decreases rapidly above 89 F, and radiation loss becomes zero when air temperature equals skin temperature.[15]

Conduction is not ordinarily an important means of heat loss. However, when large areas of the body are in contact with solid objects, conduction assumes greater importance. The use of a water-cooled mattress [10] or ice-bags are examples of the effective application of conductive heat loss. The former has the advantage of making use of a large portion of the body area that would not otherwise be available for cooling the body. Under operating room conditions, the methods that we have found most effective in treating pyrexia are those that utilize conduction.

Heat loss by convection is that heat lost by warming of the air that comes in contact with the body. Loss from natural convection is always small; however, forced convection is much more effective. Heat loss by the latter is proportional to the square root of the air velocity.[16] Surgical draping impedes convection. Moreover, the promotion of air currents in operating rooms by fans is not generally approved. As a consequence, forced convection as a means of heat dissipation is frequently unavailable to the surgical patient.

At comfortable room temperature (68 to 70 F), evaporation plays a minor role in body heat elimination; above 88 to 90 F, however, it is the principal means, and above 94 F it is the sole means by which body heat can be lost.[15] It can be seen from table 4 that above a room temperature of 85 F, the temperature under the surgical drapes is 96 F or more. Hence, above this room temperature, it is clear that in any patient in whom the sweating mechanism fails heat retention will almost certainly develop. It is our opinion that the lightest possible draping should be used during hot weather. Asepsis is actually maintained more satisfactorily with a light drape, for the patient is less likely to sweat through and contaminate it.

TABLE 5.—*Average Fahrenheit Temperatures in Various Locations in To-and-Fro, Circle, and Non-Rebreathing Anesthetic Systems*

	Location				
	Bag	Canister	Adaptor	Mid-Tube	Low Trachea
To-and-Fro System					
No. of measurements...	175	251	996	488	1,268
No. of cases.....	7	22	33	13	37
Variation, F	80.0-96.2	80.4-141.8	98.0-122.8	97.4-111.0	98.2-108.0
Mean temperature, F...	86.6	116.2	105.8	103.1	101.8
Circle System					
No. of measurements...	189	57	943	128	1,479
No. of cases.....	7	3	27	4	41
Variation, F	80.8-96.0	75.2-137.2	86.2-99.8	95.6-100.4	93.6-101.2
Mean temperature, F...	86.9	117.5	91.7	97.9	96.9
Non-Rebreathing System					
No. of measurements...	175	...	604	...	500
No. of cases.....	5	...	16	...	16
Variation, F	75.0-88.0	...	84.2-94.6	...	90.0-99.4
Mean temperature, F...	81.1	...	89.7	...	94.1

TABLE 6.—*Temperature Within the Accordion Tubing in the Circle System*

	Inspiratory Tubing		Expiratory Tubing	
	Adjoining Machine	Adjoining Y Piece	Adjoining Machine	Adjoining Y Piece
No. of measurements.....	159	159	39	39
No. of cases.....	6	6	2	2
Mean temperature, F.....	90.8	89.4	87.0	91.2

15. Hardy, J. D.; Milhorat, A. T., and DuBois, E. F.: Basal Metabolism and Heat Loss of Young Women at Temperatures from 22° C. to 35° C., J. Nutrition **21**: 383, 1941.

16. Machle, W., and Hatch, T. F.: Heat; Man's Exchanges and Physiological Responses, Physiol. Rev. **27**: 200, 1947.

Water loss through the skin includes that which is transudated as well as that secreted by the sweat glands. On exposure to heat, the rate of transudation may rise to 50 ml. per square meter per hour or only about 10% of the output of the sweat glands under similar conditions. However, evaporation of transudates assumes importance when the sweating mechanism fails, e. g., heat retention, under conditions in which evaporation is the only means of heat elimination.

Suppression of sweating was uniformly observed before the onset of heat retention in all 10 cases in which it occurred during this study. This has been noted by others.[1] Heat retention was marked not only by the height to which body temperature rose but by its progressive and rapid rise. A rate of temperature rise of 1.2 to 2.0 degrees F per hour was observed at some time during the development of heat retention in all cases.

In some of the patients in whom pyrexia occurred, concomitant measurements of the posterior nasal temperature were made with a mercury clinical thermometer. This temperature was found to provide a very adequate means of detecting and following the course of heat retention. A rectal type thermometer is recommended to avoid the risk of breakage in the nose, and it is advised that the thermometer be inserted at least 2½ in. (6.25 cm.).

The key relationship upon which evaporative heat loss depends is the difference between the vapor pressure of water at skin temperature and the vapor pressure of water in room air. (The latter is equal to the vapor pressure of water at air temperature multiplied by the relative humidity.) When this difference becomes zero at wet-bulb temperatures above 96 F, heat loss by evaporation ceases. A fairly close parallelism exists between wet-bulb temperature and the vapor pressure of water at the wet-bulb temperature. For this reason, the wet-bulb temperature is the most important factor in determining the upper limits of tolerability of an environment, as has been shown by Eichna and associates.[17]

The dry-bulb temperature taken alone is a poor index of the thermal tolerability of an operating room. A comparison of figures 3 and 4 will make this clear. Both show body temperature changes occurring in relation to similar dry-bulb temperature ranges. However, in figure 4 where the relative humidity is high, the corresponding rate of temperature rise is distinctly steeper than in the other where relative humidity is low.

The relative humidity is misleading and unsatisfactory as a measure of the tolerability of an environment because it bears no absolute relationship to the vapor pressure of water in the air; the relative humidity may be the same at two different dry-bulb temperatures, and the moisture content be very different. Figure 3 illustrates the point; one is led to expect from the three lowest charts that, if the relative humidity is below 55%, evaporative loss will be adequate to prevent marked elevations of body temperature. However, the uppermost chart reveals rises in body temperature higher than some seen in figure 4. The explanation is that, although the relative humidity is below 55% in all the charts in figure 3, the average wet-bulb temperature is 76.5 F in the top chart and only 63 to 70 F in the bottom three.

Figure 5 shows the data replotted in relation to the wet-bulb temperature only. Without respect to system, the trends of body temperature rise follow intimately corresponding rises in wet-bulb temperature. It is to be noted that, above 75 F wet-bulb temperature, body temperature rises rapidly; this effect is marked in patients anesthetized by means of the to-and-fro system, but it also occurs when circle and non-rebreathing systems are used. In 10 patients in whom heat retention occurred, the average wet-bulb temperature ranged from 74 to 82 F; in only one case was the wet-bulb temperature below 75 F.

Evaporation of water from the lung normally dissipates about 10% of the body's heat loss. Warming the respired air to body temperature accounts for an additional 1 or 2%. The fact that this avenue of heat loss is both small and self-limited should be appreciated before any discussion of the effects of anesthesia system on heat economy is undertaken. For example, the non-rebreathing system permits free lung evaporation, yet this factor alone was not enough to prevent a slow rise in body temperature at comfortable room temperatures (fig. 5). This signifies that the summation of factors impeding heat dissipation or resulting in heat gain exceeded the effect of the patient's heat dissipating mechanisms including lung evaporation.

All gases have a very low specific heat; for this reason, only small amounts of heat are involved in heating or cooling large volumes of gas. For example, the heat loss to the body in raising the temperature of 500 liters of gas per hour in the non-rebreathing system from 81 F (fig. 6) to body temperature is 1.5 Cal. per hour. (Calculations: 500 liters times 10 degrees C times 0.0003 Cal. per liter per degree centigrade [specific heat of air] equals 1.5 Cal. per hour.)

Much more important as a factor in heat exchange via the lung is evaporation. In the non-rebreathing system, anhydrous gases are inspired, and gases saturated with water vapor at about 90 F are exhaled through the valve. Seventeen grams of water per hour representing 9.8 Cal. per hour heat loss may be vaporized in such a system. (Calculation: 0.034 gm. of water per liter of saturated gas times 500 liters per hour equals 17 gm. of water per hour; 17 gm. times 0.58 Cal. per gram. [heat of vaporization] equals 9.8 Cal. per hour.) The total heat loss, 11.3 Cal. per hour in this hypothetical example, represents about 16% of the 71 Cal. per hour basal heat production of an average-sized young man. It should be noted that, of this, over five-sixths is contributed by evaporative loss.

Lesser amounts of water are evaporated from the lung when the circle system is used. This can occur because gases saturated with water vapor are exhaled into the circle system at about 8 degrees F higher temperature than they return saturated to the patient. Gases return saturated because of the water produced by the neutralization reaction between carbon dioxide and soda lime.

17. Eichna, L. W.; Ashe, W. F., Jr.; Bean, W. B., and Shelly, W. B.: The Upper Limits of Environmental Heat and Humidity Tolerated by Acclimatized Men Working in Hot Environments, J. Indust. Hyg. & Toxicol. 27: 59, 1945.

Condensation occurs in the accordion tube returning gases to the machine, which is at a lower temperature than the exhaled gas. Evaporation by this means is limited and is about 3.3 gm. of water per hour, corresponding to a heat loss of 1.9 Cal. per hour. (Calculation: the number of grams of water in 500 liters of saturated air at 97 F, the temperature of exhaled gases [0.0402 gm. per liter times 500 equals 20.1 gm.], minus the grams of water in 500 liters of saturated air at 89 F, the temperature of delivered gases [0.0335 times 500 equals 16.8 gm.] equals 3.3 gm. of water evaporated per hour. Besides this, 0.7 Cal. per hour is lost by warming gases to body temperature.) The total heat loss by the use of the circle system is about 4% of the basal heat production.

When the to-and-fro system is used, evaporation from the lung cannot occur because the temperature throughout the system is hotter than the exhaled gases (fig. 6). In fact, heat gain to the body results, not principally because of warming by hot gases, but because water condenses within the lung and airway when hot gases saturated with water vapor are cooled to body temperature. If the average temperature of inspired gases is 106 F, and it is assumed that the gas is cooled to body temperature by the time it reaches the alveoli, heat gain to the body through cooling of gases would be 0.6 Cal. per hour. On the other hand, the amount of condensation calculated to occur between lips and lung is 4.3 gm. of water per hour, equivalent to a heat gain of 2.5 Cal. per hour. (0.0532 gm. per liter times 500 liters minus 0.0442 gm. per liter times 500 liters equals 4.3 gm. of water per hour). The total heat gain represents an increase of about 4% of the basal heat production.

Cole has recently proposed that condensation of water in the respiratory tract constitutes a hazard in the use of carbon dioxide absorption systems. Such condensation does not occur when the circle absorption system is used, as is shown above, but only during the use of the to-and-fro system. The authors do not believe that this condensation per se constitutes any hazard to the patient, but may be disadvantageous only from the standpoint of the resultant heat gain to the body. At comfortable environmental temperatures, patients may be anesthetized for many hours by the absorption technique with no harmful effects whatsoever. Under these conditions, alternative avenues of heat dissipation besides evaporation from the lung are available.

On the other hand, under conditions of severe external thermal stress, i. e., wet-bulb temperatures of 75 F or higher, the heat dissipating mechanisms of the body are taxed greatly. Here, the use of the to-and-fro system, which results in a 4% heat gain rather than varying amounts of heat loss afforded by the other systems, is frequently associated with heat retention. In 9 of the 10 cases in this study in which heat retention developed, the to-and-fro system was used.

The quantitative differences in rate of body temperature rise with the three anesthesia systems are best seen in figure 5. This figure is chosen because here body temperature change is shown in relation to wet-bulb temperature, the most valid measure of the thermal stress of an environment. In figures 3 and 4, the issue of system effect is clouded by the vagaries resulting from subdivision according to dry-bulb temperature.

The height and rate of body temperature rise in patients anesthetized with the to-and-fro system were slightly but consistently greater than that seen in cases in which circle or non-rebreathing systems were used. This effect was considerably more marked above 75 F wet-bulb because of the greater number of patients in whom heat retention developed with the to-and-fro system. The reason for these effects seems to be clearly related to the differences in heat exchange via the lung that have already been discussed. Applying ice-bags to the to-and-fro cannister has been shown elsewhere [12] to reduce the air temperature effectually within the system. By this means, it may be possible to reduce the incidence of heat retention with use of the to-and-fro system to a level comparable to that of other inhalation systems.

Not as readily explained is the similarity of body temperature rises seen with the use of the circle and the non-rebreathing system. The non-rebreathing system should permit dissipation of 16% of the basal heat production, as compared with only 4% by the circle system. However, the temperature rise in certain of the charts in figure 5 is slightly higher with the non-rebreathing than with the circle system. No completely satisfactory explanation for this can be offered. It may be significant, however, that all patients anesthetized with the non-rebreathing system breathed spontaneously throughout, and respiration was not assisted. The respiration of most of the patients anesthetized with circle absorption was assisted, and in some, controlled respiration was performed. It is possible that the average respiratory minute-volume in the circle cases may have been greater than that in the non-rebreathing group. Since heat loss from the lung is directly related to the minute-volume, this may provide a partial explanation. In addition, since muscular activity increases heat production, controlled or assisted respiration should be associated with less internal heat production than when respiration is spontaneous.

Mention should be made of the treatment of heat retention in patients undergoing surgery. At the outset, the object of treatment should be clearly defined, i. e., the rapid reduction of the elevated body temperature. This is the paramount consideration, because to permit body temperature to remain markedly elevated is to risk injury to the central nervous system and liver and circulatory collapse. In experimental hyperthermia, the survival rate of animals is directly related to the speed with which the body temperature is lowered to normal limits.[6]

It should be added that it is no simple matter to prevent progression of hyperthermia in an adult of large body mass, and even more difficult to lower his body temperature to normal. The surgical draping not only impedes convection and evaporation but severely limits the body surface area available for cooling therapy. Under these circumstances, evaporative cooling methods, including the vaporization of volatile liquids on the skin, have proved ineffectual in our hands. As was mentioned

previously, under operating room conditions, the most effective methods of treating pyrexia were found to be those that utilized conduction—the water-cooled mattress and ice-bags. In treating a large group of nonsurgical patients with heat retention, Ferris and co-workers [1] also concluded that evaporative methods reduced body temperature too slowly and recommended therapy with ice.

The effectiveness of ice-bags, properly placed and used in sufficient numbers, in promptly relieving dangerous degrees of hyperthermia is clearly shown in figure 1. The preferred locations for placement of ice-bags, insofar as the surgery permits, are between the thighs, the inguinal regions, axillas, flanks, and chest. In adults, eight or more bags, frequently refilled, are often required to reverse the course of a rapidly rising body temperature. Ice-bags, though somewhat inconvenient to use effectively, have the advantage of being generally available in hospitals.

The convenience of the water-cooled mattress when available, and its effectiveness used prophylactically in children is indisputable.[10] Our experience with the mattress has been limited to its use in children, where it has proved very satisfactory. Extension of its use to adults can be recommended. Other effectual contributory methods of treating heat retention include lightening the surgical drape and promoting air currents under the drapes.

Of lesser value in treatment are the use of evaporative methods of cooling, lightening of anesthesia (to lessen depression of the central thermoregulatory center), adequate replacement of water and salt, and changing from the to-and-fro anesthesia system to one that permits dissipation of heat by the lung. These less effectual methods are supplementary and should never be relied on alone to lower marked elevations of temperature.

Heat retention in surgical patients can be prevented. Air conditioning is of course very desirable from this point of view. Where air conditioning is not available, it is advisable to postpone elective surgical procedures when the wet-bulb temperature of the operating room is 75 F or above. The results of this study indicate that above this temperature, body temperature tends to rise substantially during prolonged operations regardless of the anesthesia system. Light surgical draping of the patient is strongly recommended during warm weather. Use of the to-and-fro anesthesia system is best avoided, or if it is used, the cannister should be cooled with ice. Low doses of belladonna premedicant drugs are advisable since they decrease sweating.[18] Patients should be brought to the operating room adequately hydrated and with any salt depletion corrected; fluid losses during surgery should be carefully replaced. Frequent measurements of the patient's body temperature should be made (the posterior nasal temperature is convenient to measure and is satisfactory) in order to permit prompt recognition of heat retention in its incipiency and institution of appropriate therapy. In addition, cessation of sweating should be regarded as an urgent danger signal. The value of the water-cooled mattress in prophylaxis has already been mentioned. All of the preceding remarks apply with special force in children, in whom the factors of a small body mass, high internal heat production, and immature thermoregulatory responses make heat retention a particular hazard.

SUMMARY AND CONCLUSIONS

Temperature measurements were made in 212 patients undergoing surgery and general anesthesia. The body temperature of 164 adult patients undergoing lengthy operations was studied in relationship to environmental temperature and humidity and choice of anesthesia system. The temperature in the airway and in various locations in the to-and-fro, circle, and non-rebreathing anesthetic systems were made. The dry-bulb temperature under the surgical drapes was measured over a 20 F room temperature range.

The wet-bulb temperature is the most satisfactory measure of the thermal tolerability of an environment. Below 75 F wet-bulb temperature, heat retention is uncommon in adult subjects who are afebrile before operation. Above this temperature, body temperature rises progressively, and sometimes markedly, regardless of the anesthesia system used.

Below 75 F wet-bulb temperature, the height and rate of body temperature rise is slightly but consistently greater in patients anesthetized with the to-and-fro system than when the circle or non-rebreathing systems are used. Above this temperature heat retention is much more frequent when the to-and-fro system is used than when circle or non-rebreathing techniques are employed. The reasons for these effects are discussed.

Heat retention is usually associated with cessation of sweating under conditions where evaporation is the principal means of heat dissipation. The rate of body temperature rise during heat retention may be as high as 1.2 degrees F to 2 degrees F per hour in adults. Methods of prevention and treatment of heat retention during surgery and anesthesia are discussed.

18. Frumin, M. J., and Papper, E. M.: Inhibition of Sweating in Man by Scopolamine, Anesthesiology **12**: 627, 1951.

Common Salt.—It is certainly difficult, and perhaps impossible, to trace the earliest human use of common salt. It has been in use by civilized people since earliest recorded history. . . . Evidence has been assembled which suggests that salt came into use at the time man made the transition from a nomadic hunter-fisher, on a roasted meat and milk diet, to an agriculturist on a cereal grain and vegetable diet. The fact that herbivorous animals manifest a craving for salt by travel for long distances to salt licks, while carnivorous animals do not, intrigued the physiologic chemists of the latter half of the nineteenth century. Bunge may have been the first to advance the concept that the high potassium content of a grain and vegetable diet required in the herbivora an increased sodium intake. . . . Whether this is the true reason agricultural man began adding salt to his diet, certainly once he had, he took a great fancy to it. The preservative effect of salt must have been noted early, and food so preserved became vital through the non-harvest seasons and during voyages and migrations. There are many references to salt in the Bible, and there is a rich and entertaining literature concerned with the history, symbolism and financial problems of salt. . . .—George R. Meneely, M.D.; Robert G. Tucker, Ph.D.; William J. Darby, M.D., Ph.D., and Stewart H. Auerbach, M.D., Chronic Sodium Chloride Toxicity: Hypertension, Renal and Vascular Lesions, *Annals of Internal Medicine*, November, 1953.

POSTOPERATIVE PSEUDOMEMBRANOUS ENTEROCOLITIS

SUCCESSFUL TREATMENT WITH CORTICOTROPIN (ACTH)

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Perhaps no other postoperative complication presents so serious and baffling a problem as pseudomembranous enterocolitis. The lack of experience with this infrequent disease makes diagnosis difficult, and the heretofore absence of known treatment makes death certain in a large number of cases. These provocative factors and our observation that the lives of these patients may be saved by brief administration of corticotropin (ACTH) testify to the importance and urgency of this problem.

Pseudomembranous enterocolitis is a clinical entity that has been observed after major surgical operations. In our series of seven cases, it occurred four times after resection of the sigmoid or sigmoid and rectum, once after vagotomy and gastrojejunostomy, once after simple cholecystectomy, and once after right hemicolectomy and resection of the terminal ileum. Even though it occurs more frequently after intestinal surgery, it has been observed after operations on the brain, breast, uterus, gallbladder, and stomach.[1] Penner and Bernheim [2] and also Dixon and Weisman [1] have described the clinical and pathological aspects of this disease. Recently pseudomembranous colitis was observed after the administration of chlortetracycline (Aureomycin) and chloramphenicol.[3]

In the past two years, we have seen three patients in whom pseudomembranous enterocolitis developed after surgery on the colon. These patients received phthalylsulfathiazole (Sulfathalidine) and oxytetracycline (Terramycin) preoperatively. After surgery they became gravely ill. Because of our impression that some intra-abdominal complication had developed, two of these patients were operated on again. At operation, the diagnosis of pseudomembranous enterocolitis was definitely established. All three patients recovered rapidly a few days after therapy with corticotropin. Four other patients are included who received intensive supportive therapy but no corticotropin. In these four, the diagnosis of pseudomembranous enterocolitis was made at necropsy.

REPORT OF CASES

CASE 1.—A white man, 73 years of age, was seen in May, 1951, with a history of intermittent rectal bleeding for one year. He was in good physical condition, except for a mild hypertension of 180/76 mm. Hg. His history was noncontributory. Rectal and sigmoidoscopic examinations revealed a small carcinoma situated 8 cm. from the anus. The mucosa of the rectosigmoid otherwise appeared normal throughout the distance of sigmoidoscopic examination. The roentgen examination of the colon was also normal in all other respects. A biopsy of the lesion revealed it to be a papilliferous adenocarcinoma. The blood cell count, urinalysis, serology, serum urea nitrogen, serum chloride, serum proteins, and albumin-globulin ratio were all normal.

Before surgery, the patient received 1.5 gm. of phthalylsulfathiazole four times daily for three days and 250 mg. of oxytetracycline every six hours for five days. After abdominoperineal resection of the rectum, the patient was given 400,000 units of penicillin every 12 hours and 500 mg. of dihydrostreptomycin every 6 hours. He received 75 mg. of vitamin K during the period of antibiotic therapy. The patient began to run a septic fever from the seventh postoperative day until corticotropin was administered on the 35th postoperative day. His pulse rate was rarely over 100 per minute. He did not have a bowel movement until the seventh postoperative day, and thereafter the stool became liquid and contained shreds of mucosa. He appeared toxic and weak; his abdomen became distended; and finally edema developed in both lower extremities and scrotum. Ascites was noted on the 16th postoperative day. The leukocyte count gradually rose to 33,900 per cubic millimeter. During this period, the patient complained of pain and tenderness over the right side of the abdomen. Repeated roentgen examination of the abdomen showed increasing elevation of the right side of the diaphragm and marked gaseous distention of the colon (fig. 1). Numerous stool examinations, including cultures, failed to reveal the presence of pathogens.

An exploratory laparotomy was performed on the 21st postoperative day; 3,000 cc. of straw-colored ascitic fluid was aspirated. All of the gross pathological changes were limited to the colon, extending from the ileocecal valve to the colostomy. The wall of the colon was markedly thickened, hyperemic, edematous, and inflamed. The lumen was greatly distended. There was no evidence of obstruction. The abdomen was closed because no other abnormality was found. The patient improved for a few days, and then septic fever, ascites, and liquid stools containing shreds of mucus and blood developed again.

Roentgen examination, with colloidal thorium dioxide (Umbrathor) as the opaque medium, revealed a markedly dilated colon with large ulcerations (fig. 2). A sigmoidoscope inserted through the colostomy to a distance of 20 cm. showed edematous, friable, and necrotic mucosa resembling, but not pathognomonic of, ulcerative colitis. The sigmoidoscopic appearance of the mucosa resembled, somewhat, the type of colitis seen in heavy metal poisoning.

In spite of vigorous supportive therapy, the patient's condition deteriorated. After considerable deliberation, he was given 20 mg. of corticotropin every six hours. Within 24 hours after initiation of this treatment, he became afebrile. The patient improved rapidly, and his appetite and a feeling of well-being returned. His stool became formed; edema and ascites disappeared. One week later, the patient was discharged. He has been well to date.

CASE 2.—A white man, 59 years of age, was admitted to the Chicago Memorial Hospital on Dec. 5, 1952. A preoperative diagnosis of papilliferous mucinous adenocarcinoma of the rectosigmoid was made from biopsy taken at sigmoidoscopy. The only other abnormal finding was a moderate hypertension of 170/100 mm. Hg. This patient received similar preoperative preparation with phthalylsulfathiazole and oxytetracycline as in case 1. In this man too, a fever ranging from 100 to 103 F developed on the first day after abdomino-

1. Dixon, C. F., and Weisman, R. E.: Acute Pseudomembranous Enteritis or Enterocolitis: Complication Following Intestinal Surgery, S. Clin. North America **28**: 999-1023 (Aug.) 1948.

2. Penner, A., and Bernheim, A. I.: Acute Postoperative Enterocolitis: Study on Pathologic Nature of Shock, Arch. Path. **27**: 966-983 (June) 1939.

3. Reiner, L.; Schlesinger, M. J., and Miller, G. M.: Pseudomembranous Colitis Following Aureomycin and Chloramphenicol, A. M. A. Arch. Path. **54**: 39-67 (July) 1952.

perineal resection of the rectum and sigmoid. His postoperative symptoms of fever, leukocytosis, diarrhea, abdominal tenderness, and lethargy duplicated, in many respects, the course described in case 1. Thirty days after surgery, the patient was given 20 mg. of corticotropin every six hours for five days. His recovery was immediate, spectacular, and complete.

Fig. 1.—Roentgenogram of patient in case 1 showing a marked gaseous distention of the colon. Gas present in several loops of small bowel resembles paralytic ileus; patient had diarrhea.

CASE 3.—A white man, aged 55, was admitted to the Chicago Memorial Hospital on May 6, 1953. A preoperative diagnosis of benign pedunculated polyp was made by roentgen examination of the colon. Sigmoidoscopic examination revealed normal rectal and sigmoidal mucosa. A moderate hypertension of 170/110 mm. Hg was noted. After preoperative preparation with phthalylsulfathiazole and oxytetracycline,

Fig. 2.—Roentgenogram showing edema and ulcerations of the colon mucosa, after colon of patient in case 1 was filled with colloidal thorium dioxide.

a sleeve resection of 20 cm. of the sigmoid colon containing the benign polyp was performed. The continuity of the sigmoid colon was reestablished by an end-to-end anastomosis. After the operation, the patient was given the usual doses of penicillin and dihydrostreptomycin. As in the other cases, the postoperative course was marked by fever, leukocytosis, abdominal tenderness, paralytic ileus, bloody diarrhea, and weakness. The blood pressure remained normal. The patient was explored on the 10th postoperative day. The only pathological change found was limited to two lesions in the distal third of the ileum. These lesions measured 3 and 5 cm. in length and consisted of edema and hyperemia of the bowel wall simulating acute regional ileitis (fig. 3). This patient had no ascites. The colon and small bowel were moderately distended with gas, and the wall of the colon was slightly edematous. Intramuscular injections of 20 units of corticotropin given every eight hours for three days were initiated on the second day after exploration. The patient made an immediate improvement; his temperature returned to normal. The administration of corticotropin was discontinued on the fourth day because psychic episodes and hallucinations develeped. The patient gradually improved and was discharged as normal on the 23rd hospital day.

The remaining four patients received no corticotropin. The diagnosis of pseudomembranous enteritis or colitis was made only at postmortem examination. The salient features of these cases are summarized in the table.

Fig. 3.—Roentgenogram of patient in case 3 showing pattern identical to that seen in paralytic ileus; patient had two small areas of necrotizing ileitis.

COMMENT

The symptoms and clinical findings common to all seven patients were fever, leukocytosis, diarrhea, weakness, lethargy, abdominal pain, tenderness, gaseous distention, and paralytic ileus. We observed shock in only three of the seven patients. In the remaining, the blood pressures during operation and hospitalization showed no significant changes. The three patients whose blood pressures fell to 80 mm. Hg systolic, or below, responded better to intravenous infusion of isotonic sodium chloride solution than to whole blood transfusion. The shock levels did not develop until the third postoperative day. This infrequent observation of shock differs somewhat from the report of Penner and Bernheim.[2] In 1939, they reported 40 cases of ulcerative enteritis found at necropsy. They concluded that the vasomotor mechanisms active in shock were responsible for the condition

called postoperative enterocolitis. The common finding in all the cases they reported was shock. In 1948, Dixon and Weisman [1] reported on 23 cases of pseudomembranous ileocolitis. Their series included 21 deaths (91% mortality). In all of their cases the disease followed operations on the gastrointestinal tract.

Most authors have been chiefly concerned with the pathological nature of the disease.[4] The real cause of this complication is unknown. A recent case report presented at the Massachusetts General Hospital [5] expressed a very plausible warning. It stated that certain antibiotics may so alter the bacterial flora of the intestine that an overgrowth of bacteria, notably staphylococci, may achieve pathogenic proportions. The patient in the case under discussion died of pseudomembranous, necrotizing enteritis. A culture of the material found in the small bowel and blood culture grew staphylococci at necropsy. Even though this may be a real complication of antibiotic therapy in some cases, it does not explain the cause of pseudomembranous or necrotizing enterocolitis. Dixon's series and our cases included exhaustive cultures of stools, but no pathogen could be incriminated as the causative agent of the disease, except in our case 10 (see table). In this instance staphylococci were cultured from the intestinal content taken at necropsy. Yet, this is an important consideration in view of our treatment of postoperative necrotizing enterocolitis with corticotropin. The patient with staphylococcic enteritis would be harmed if given corticotropin; therefore, diagnosis of pseudomembranous enterocolitis must be made accurately and bacterial enteritis ruled out by careful stool cultures, before the use of corticotropin.

The assumption that pseudomembranous enteritis and colitis are associated with the preoperative use of antibiotics is questionable. The first case was described by Finney [6] in 1893; certainly his patient received no antibiotics. The same is true of the series described by Penner and Bernheim in 1939. Three of our patients received no antibiotics preoperatively. These positive facts are strong evidence against the causative role of the antibiotics.

Cognizant of the importance of shock in pseudomembranous enterocolitis, Hilsabeck and Dixon [7] stressed vigorous antishock treatment. Since we do not know the cause of the disease, we may indulge in reasonable speculations. Shock in this case may, at least in part, be due to loss of fluids and electrolytes by way of diarrheal stools. The treatment, therefore, must consist of vigorous replacement of water and electrolytes. Be that as it may, it was not possible to keep four of our patients alive by vigorous water and electrolyte replacement, supplemented by plasma and blood transfusions. In the consideration of the cause of necrotizing enteritis and colitis, one must, therefore, realize that measurable loss of water and electrolytes when adequately replaced, still do not prevent death from shock. It must be reemphasized that clinically recognizable shock did not occur in all our cases. We did not observe excessive blood loss or infection or cardiac involvement prior to or shortly after surgery that could be held responsible for peripheral vascular collapse. Innumerable surgical or hemorrhagic shocks and peripheral vascular collapse in medical diseases have, on the other hand, been observed in the past without the manifestation of necrotizing enterocolitis. Shock, therefore, played no major role in the cause of the disease in our experience.

After observing four deaths, we decided to treat this fatal complication with adrenocorticotrophic hormone. The three patients who were treated with corticotropin recovered promptly and completely. Even though the series of cases is small, we offer this method as a lifesaving therapy for pseudomembranous enterocolitis, a complication that up to the present time was almost always fatal.

The strikingly beneficial effect of the therapy with corticotropin in our cases permits further speculation as to the mechanisms of this disease process. Any bacterial disease would have been markedly aggravated by corticotropin. Septicemia or spread of the local infection would be the expected result of such therapy. We believe, therefore, that the disease is not an infection; likewise, it cannot be assumed that the disease is of endocrine origin. It has been well established that the pharmacological action of adrenal steroids stimulated by corticotropin becomes evident in a variety of conditions other than

Summary of Four Cases of Pseudomembranous Enteritis or Colitis Diagnosed at Autopsy

Case*	Age, Yr.	Preoperative Medication	Diagnosis and Operation	Onset of Diarrhea, Postoperative Day	Corticotropin (ACTH)	Results
4	56	Phthalylsulfathiazole and oxytetracycline	Ulcerative colitis with old ileostomy; total colectomy; combined abdominoperineal resection	3rd	None	Died; necropsy—pseudomembranous enteritis
5	44	None	Cholecystitis and cholelithiasis; cholecystectomy; explored	4th	None	Died; necropsy—pseudomembranous enteritis
6†	54	None	Duodenal ulcer; vagotomy; gastrojejunostomy	4th	None	Died; necropsy—acute necrotizing ileitis
7‡	29	None	Terminal ileitis; right hemicolectomy; terminal ileum	None	None	Died; severe enteritis

* Patients were all men.
† Data on patient in case 6 supplied by Dr. Hilger P. Jenkins.
‡ Data on patient in case 7 supplied by Dr. Samuel L. Goldberg.

4. Kleckner, M. S., Jr.; Bargen, J. A., and Baggenstoss, A. H.: Acute Pseudomembranous Enterocolitis, Proc. Staff Meet., Mayo Clin. **28**: 313-319 (June 3) 1953. Penner and Bernheim.[3] Reiner and others.[3]

5. Case Records of the Massachusetts General Hospital, Case 39271, New England J. Med. **249**: 29-33 (July 2) 1953.

6. Finney, J. M. T.: Gastroenterostomy for Cicatrizing Ulcer of Pylorus, Bull. Johns Hopkins Hosp. **4**: 53-54 (March) 1893.

7. Hilsabeck, J. R., and Dixon, C. F.: Treatment of Acute Postoperative Pseudomembranous Enterocolitis, Am. J. Surg. **84**: 114-117 (July) 1952.

endocrine in nature. Furthermore, the pathological changes in the gastrointestinal tract in cases of proved adrenal failure do not include changes suggesting pseudomembranous enterocolitis. Experimental studies are in progress to elucidate the mechanism of this condition. One of the possibilities being explored is that of tissue necrosis resulting from antigen-antibody reaction. Further clinical observations and experimental work will be necessary to prove such an assumption.

SUMMARY AND CONCLUSIONS

Acute, postoperative pseudomembranous enterocolitis was cured in three patients with the administration of 20 mg. of corticotropin (ACTH) three times daily for periods ranging from 4 to 10 days. Bacterial enteritis should be ruled out before the administration of corticotropin. The possible causes of the disease are discussed, but, as yet, the true cause remains unknown.

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RADIATION MANAGEMENT OF OTHERWISE HOPELESS THORACIC NEOPLASMS

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and
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Every physician should be aware of his responsibility if he declares a cancer patient hopeless and leaves him to his fate. The term "hopeless" implies the death sentence of the individual patient. In medical use this term can mean either incurability or no possibility of a favorable influence on the patient's condition, even of a palliative nature. In our interpretation, the term "hopeless" should be reserved for the very few patients whose suffering might be increased or whose fate might be speeded by any method of attacking the cancer. These are obviously terminal situations.

"Hopeless" is usually applied when a thoracic neoplasm is inoperable, although we often see patients with lesions considered operable who have no chance of cure by surgery, owing to spread of the neoplasm. Further, we know that some surgically hopeless patients can be cured by radiation, and that good palliation can be obtained in excess of that resulting from other known forms of treatment. The secret of increasing improvement in these situations is giving more patients the benefit of the doubt and giving a carefully planned treatment with adequate dosage. In the older material that we have reviewed there are only a few cases in which a palliative gesture resulted in a surprising degree of response, but these treatments had been performed without a uniform plan.

Our attitude toward palliation and curability has been influenced by experiences with the 23 mev betatron. In experimental work with it we have been impressed with its effects on local or primary neoplasms even though fatal invasion of the disease was in progress elsewhere. Consequently, our feeling is that we could offer better results if patients with cancer were referred to us earlier. Our continuing use of conventional radiation therapy has been enhanced as a result of transfer of ideas and methods from betatron treatment to the more readily available machines.

What should be regarded as accomplishment in palliative effect? The usual criterion of five year survival cannot be used, nor does absolute survival time alone reflect the true nature of palliative effect. If one follows with full human sympathy the fate and long suffering of these unhappy patients, one cannot appraise highly enough the temporary relief enabling them to return to a useful family, social, and professional life for any length of time. Survival time may not be significantly prolonged in many of these patients, but the important thing is that terminal suffering is frequently postponed and shortened by these palliative effects. Therefore, we consider the following factors in evaluating palliation: 1. The objective local effect. This is judged by physical examinations, by comparative measurements on the surface, by palpation, by roentgenograms, and by histopathology either serially during life or by final comparison at autopsy. 2. The clinical symptoms. Such criteria as pain, cough, dyspnea, and dysphagia are used and are evaluated in terms of effort expended, medication required, and changes in habits or preferences. These impressions are best gained from the patient, his family, and the physician. 3. The general condition. This relates to weight, strength, working ability, psychological attitude to normal family and social life, and duration of useful life. 4. The absolute survival time from start of radiation. 5. The extent of terminal distress. This is evaluated in terms of postponement, duration as compared with survival time, and intensity.

The clinical material that we wish to present represents some of our experiences and impressions from working with hopeless chest tumors, primarily orginating in the lung and esophagus. The selectively bad composition of our material, the varying locations of primary tumors, the differences in histopathology, and the spread in age incidence continue to hamper our statistical inclinations; therefore, we will refer to representative patients treated with the betatron and others treated by conventional x-rays.

BETATRON X-RAY TREATMENT

The lung cancer cases can be separated into two groups in which the results were very favorable: Pancoast tumors and primary lesions of the upper bronchus. Four patients suffering from the Pancoast type of tumor have been treated. One is living and well 34 months after

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Lack of space limits the number of figures to six representative cases instead of the original 23 cases.

irradiation and is working daily. The original intractable pain gradually disappeared during the course of treatment and the original rib destruction spontaneously recalcified (fig. 1*A* and 1*B*), but radiation fibrosis augmented by excessive dosage and too rapidly given treatment during our early experience has produced a fluctuating circulatory disturbance. This may have been

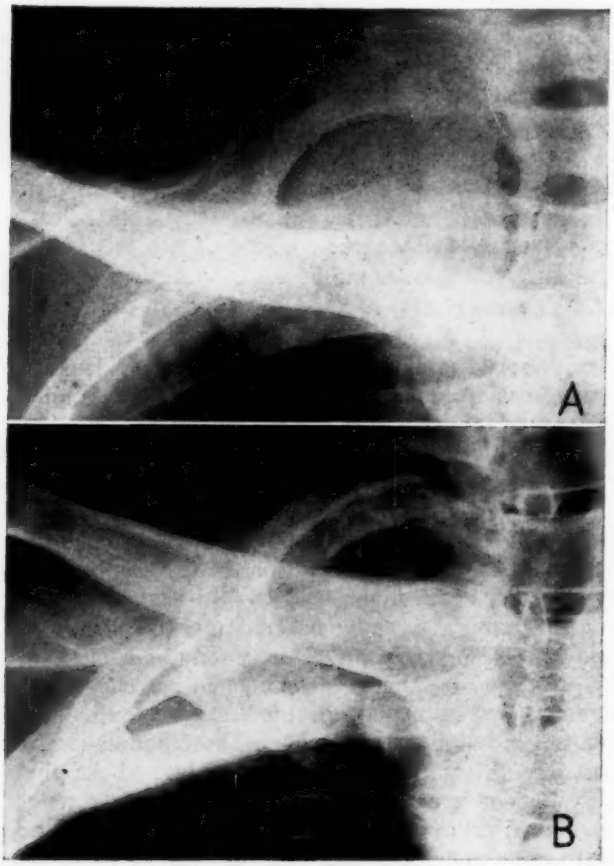

Fig. 1. *A*, tumor of the right superior sulcus, with destruction of the second rib. *B*, two years after betatron irradiation (11,000 "r" tumor dose in 26 days, eight fields). Recalcification of the second rib. After 35 months the patient was living, without tumor evidence.

further aggravated by a series of post-treatment biopsies, all of which showed only fibrosis. A second patient followed an almost identical course of relief of pain, reconstitution of destroyed ribs,[1] and working ability for 17½ months, at which time he died at home. Information from relatives was not conclusive enough to indicate coronary occlusion or recurrence of tumor. A third patient has been observed for eight months and is also asymptomatic and working. The destroyed bony structures are recalcifying. A fourth patient is under treatment and is already markedly relieved of pain.

In the second group are upper bronchus lesions with superior-anterior mediastinal metastases. One patient had a right-sided primary lesion with peripheral subpleural metastases and large cervical node masses that caused choking and superior vena cava symptoms. Betatron x-rays were used, and after this there was marked shrinkage of areas of involvement, respiratory and circulatory symptoms disappeared, and the agony of a death from vena cava syndrome was averted.[2] Metastases in the abdomen and liver, however, subsequently led to a rapid decline. The second patient, with a primary tumor in the upper left bronchus, is still living, free from pain, working, and without radiological evidence of tumor 16 months after treatment (fig. 2*A* and 2*B*).

A third group of nine patients, with varying localizations and extensions, did not have a significantly increased survival time, but palliation could be noted in the majority by any standards. In several patients local growth arrest or regression of the primary tumor could be observed on roentgenograms. In one patient a terminal manifestation of extensive bilateral lung metastases was limited to the lung areas that had not been irradiated.

The most gratifying effect of this treatment is measured in terms of clinical symptoms. Most patients were definitely improved. Pain, dyspnea, cough, and expectoration frequently subsided or diminished. Some patients regained their strength and their hope in life and resumed their usual useful way of life. A cheerful outlook is not maintained for very long, however, since in most of these patients there is a rather sudden and rapid downhill course. The terminal agony is therefore comparatively shortened, in some cases to only one week. In this connection two patients should be mentioned. One was given irradiation eight months after diagnosis, during which time he had become incapacitated. After he was given irradiation he lived six and one-half months without pain, and with only trivial cough and expectoration. He was able to work up until a week before his death, when sudden occlusion of a main bronchus occurred. The other patient was disabled by a lower right bronchus lesion. Exploration showed tumor invasion of the lower pulmonary vein and right auricular wall. A known metastasis in the iliac spine prevented walking. After irradiation was given to the chest and iliac lesions, the chest pain disappeared, the cough subsided, the lung lesion regressed, and the iliac metastatic area began to recalcify. The patient could walk unaided. As a physician he was able to resume practice and continued for three and one-half months until a week before death, when dyspnea occurred suddenly and progressed rapidly. Radiological regression in this patient is shown in figure 3.

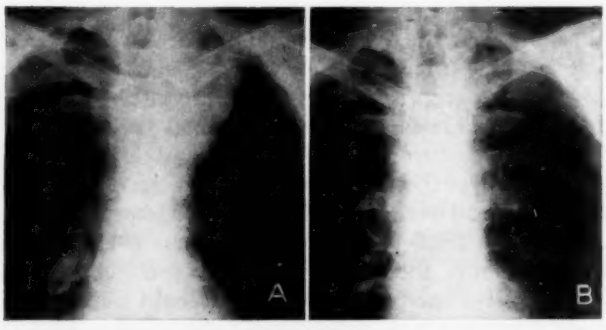

Fig. 2. *A*, bronchogenic carcinoma of the left upper lobe with metastatic mass in the anterior upper mediastinum, extending to the right. Paralysis of the left vocal cord. *B*, seven months after betatron irradiation (10,000 "r" tumor dose in 40 days, six fields). The tumor mass disappeared. After 16 months the patient was living, without evidence of tumor.

In trying to explain why Pancoast tumors and the more superior-anterior lesions seem to respond better, we

1. Harvey, R. A.; Haas, L. L., and Laughlin, J. S.: Betatron Cancer Therapy, Radiology **58**: 23-34, 1952, fig. 7 and 8.
2. Harvey, R. A., and others,[1] fig 10 and 11.

must take into account earlier diagnosis and the relatively late incidence of lymphatic and hematogenous spreads in these cases. This permits a greater chance of local annihilation of the lesion by irradiation. In other locations the spread seems to be faster, is especially common by the time our patients are given diagnoses and is almost uniform by the time they are referred for treatment. We feel that the effort expended in treating these patients is worth while and would promise better final results if the radiation could be applied to more favorable stages of bronchogenic cancer.

Not much could be expected in the way of results in our patients with esophageal cancer because they were all in the most advanced stages. These patients do permit the development of techniques, however, and as we can destroy more and more areas of involvement, we develop courage to take the responsibility of attempting cure in more favorable cases. All of these patients have had either distant metastases or involvement of vessels, lymphatics, trachea, etc. It is no wonder that of six patients in this group, only one survived 7 months, and one 11 months. Additional recent patients are still living. One of the reasons why we see these patients so late is that so much time is lost between symptoms and diagnosis and further between diagnosis and radiation while various other treatments are considered and abandoned. The significance of delay is demonstrated in one of our patients. There was only a submucous constricting ring at initial diagnosis. Seventeen days later the tumor broke through, formed a fungating mass, and occluded the lumen (fig. 4*A* and 4*B*).

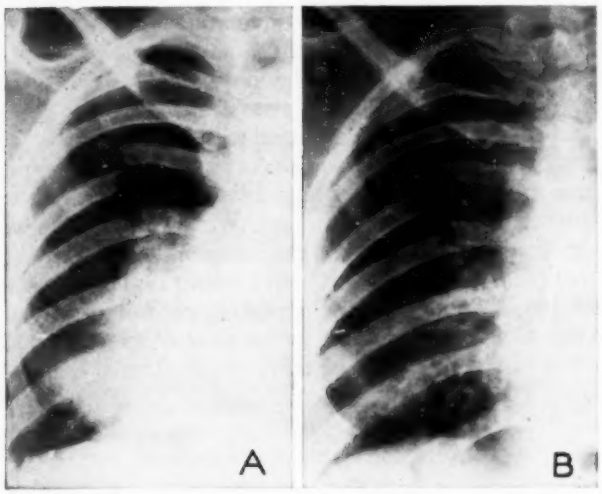

Fig. 3. *A*, bronchogenic, poorly differentiated epidermoid carcinoma. Multiple metastases were already present at time of the irradiation. *B*, after palliative betatron irradiation (8,500 "r" in 38 days). Radiological regression of the primary lesion and clinical regression of the pulmonary symptoms. The patient died in two and one-half months with generalized metastases.

In spite of the short survival of the patients, our experiences are very encouraging and raise the hope that esophageal carcinoma patients can be given irradiation with much better results than thought possible in the past. Nearly all of these patients showed marked improvement in the mechanical function of the involved area, and some lesions appeared to heal. In two patients autopsies showed no gross or microscopic evidence of residual cancer in the treated zone. No significant difference in palliation appeared in this small group among cervical, middle, or lower esophagus lesions. An increase in the caliber of the passage was noted in most patients, and some had a normal lumen restored (fig. 5*A* and 5*B*, 6*A* and 6*B*). In spite of this, we urge gastrostomy in every case. In some of the cases in which this was resisted, either by the referring physician or by the patient, it had to be performed during, or after, treatment. This necessarily interrupted treatment and further aggravated the weakened condition of the patient at a critical time. There are several reasons for requiring a gastrostomy in these patients. 1. Malnutrition and even cachexia are already present in most of these patients, and this cannot be satisfactorily overcome while a progressing disturbance of the esophagus exists. 2. Delivery of an adequate dosage of radiation produces a mucosal reaction in the membrane for several days or weeks that adds to the discom-

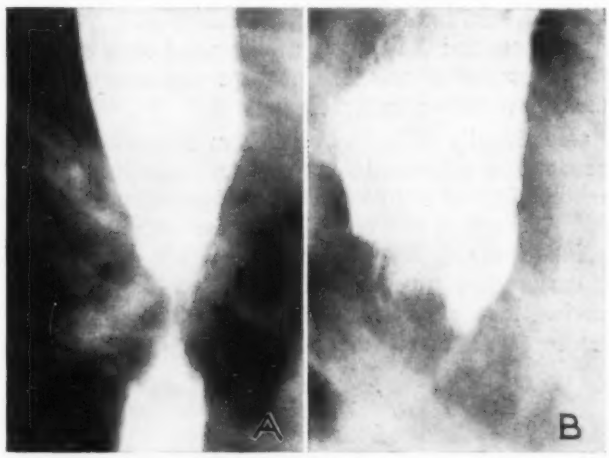

Fig. 4. *A*, epidermoid carcinoma of the esophagus; only annular constriction. *B*, seventeen days later, fungating tumor mass occluding the lumen with suprastenotic dilatation.

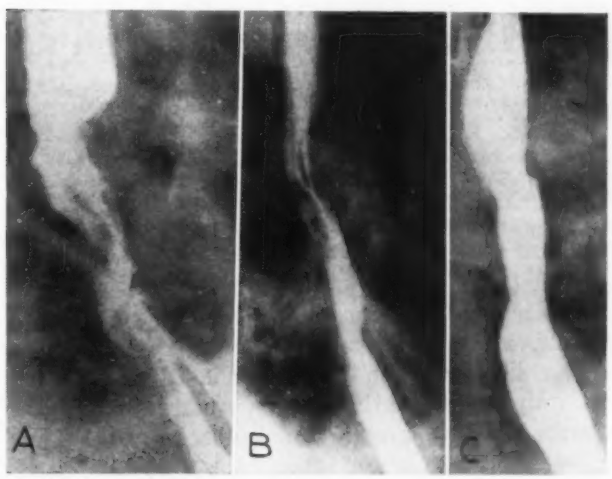

Fig. 5. *A*, epidermoid carcinoma of the esophagus with moderate prestenotic dilatation, before irradiation. *B*, after betatron irradiation (10,000 "r" in 4½ weeks). Normal contours and folds, free passage. *C*, five months later, generally wider lumen, but in the central portion rigid wall. In spite of the local regression, constant dysphagia, gradual weakening of the patient. The patient died in five and one-half months of bronchopneumonia. Autopsy revealed no residual tumor cells. The mucosal surface showed a denuded, nonreepithelized area in the scarred rigid segment. Lungs showed irradiation fibrosis.

fort of swallowing and undernutrition. 3. The breakdown and disappearance of tumor tissue leaves a temporarily denuded esophageal wall without mucosal covering and, at times, with a slow healing ulcerative-like defect in the zone of the original lesion (fig. 6*C*). These reepithelize slowly, and the denuded area is particularly vulnerable. It continues the symptoms of dysphagia, and solid food may cause reulceration or elicit hemorrhage. 4. The healing process and scarring of the denuded area increases the chances of additional shrinkage and narrowing for weeks or months after irradiation.

Occasionally difficulties arise from gastrostomies, but these can be minimized by proper postoperative care and training. One of our patients died of postoperative peritonitis. Hospital bed facilities for the temporary postradiative care of these patients is, at times, as important as the postoperative hospital care. Both palliation and survival time would reflect the advantage of adequate nursing care of these patients after radiation.

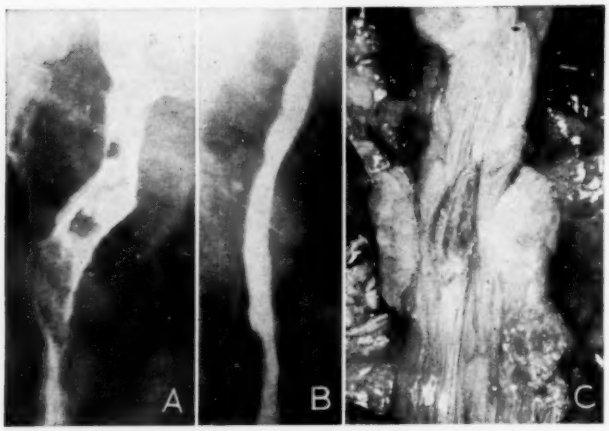

Fig. 6. *A*, extensive epidermoid carcinoma of the esophagus. *B*, after betatron irradiation of 10,000 "r" in 29 days, regression of the lesion, free passage; probably ulceration of the mucosa. *C*, autopsy after three months showed generalized metastases, but no residual cells locally. A nonreepithelized, denuded area at the mucosal surface. Periesophageal mass at the ulcer level was histologically fibrous scar tissue and contained no more tumor cells.

CONVENTIONAL X-RAY TREATMENT

In the last 15 years a large number of similar patients have been treated in our department with 200 kv. and 400 kv. x-rays. Statistical comparisons between this group and the betatron group are not significant at this time owing to the marked disproportion in numbers of patients in the two groups and also because of the poor follow-up in many of the earlier cases. Palliative effects were, and continue to be, observed in patients treated with conventional x-rays, but our present impression is that it is not so consistently observed or so prominent in this group as in the betatron group. Survival has rarely continued as long as six months, and we have not seen a complete cancerocidal effect in autopsy specimens from this group.

During the last year seven patients with bronchogenic cancer were treated with the aid of grid techniques. Moderate palliation was obtained in two of them, but none survived six months. Among the patients treated without grid, palliation was spotty. One patient resumed work temporarily and survived eight months. Another is alive and in good condition over a year later but is unusual in that the tumor was so radiosensitive. He received only 1,100 r tumor dose without grid and 1,100 r with grid. His biopsy specimen has obviously been reevaluated several times but without change in opinion regarding the diagnosis.

The esophageal cancer patients showed better palliation during this same period. Three patients have had excellent palliation: one is in good condition 14 months later, but with metastatic cancer in the lymph nodes of the neck; another had complete relief of obstructive symptoms for 4 months, and recurrent symptoms at a different level disappeared for 5 months after another course of treatment. An additional patient is doing very well 8 months after treatment.

COMMENT

Any difference in results between the betatron group and the conventional x-ray group is undoubtedly due to a more adequate and uniform depth dose distribution with the betatron. In spite of this, palliation can also be expected to follow adequate irradiation with conventional x-ray therapy. Therefore, no patient should be denied irradiation therapy unless he has reached a terminal status. High voltage therapy is preferable if it is available, but a good effect can be obtained with conventional x-ray therapy if the therapist will devote just as much care to these patients with a poor prognosis as he would to those patients with a chance of cure. We rarely distinguish, with the betatron, between palliative and curative treatment planning. We give as high a tumor dose as possible and judge it primarily by individual tolerance. One can make a good orientation as to patient tolerance and tumor susceptibility on the basis of a half curative tumor dose (3,000 r at 400 kv., equivalent to 5,000 r with the betatron).

SUMMARY AND CONCLUSIONS

The term "hopeless" should not be applied to any cancer patient until all known therapeutic procedures have been attempted or the patient is in a terminal status. "Hopeless" should not be used just because one particular method of treatment offers no chance of cure or good palliation.

Palliation in thoracic neoplasms is judged on the basis of objective local effects, alteration in clinical symptoms, general condition and useful life of the patient, absolute survival time, and the duration of the terminal distress. The 23 mev betatron x-ray beam shows unusual evidence of palliation and possibility of cure for patients with the Pancoast type of tumor of the upper chest, better than average palliation with commoner advanced bronchogenic cancers, and good palliation with indications of cures possible for primary esophageal neoplasms in earlier stages. Conventional x-ray treatments also offer palliation, but so far they offer less chance for patients with advanced cancer, owing to less adequate and uniform tumor dose. High voltage therapy planning does not distinguish between palliative and curative intent. If the same care were used with conventional x-ray treatment planning, greater palliation for patients with advanced cancer would be obtained.

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CLINICAL NOTES

TREATMENT OF ACUTE HEROIN INTOXICATION WITH NALORPHINE (NALLINE) HYDROCHLORIDE

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Numerous reports have appeared in the literature demonstrating that in animals nalorphine (Nalline) hydrochloride is an effective antagonist to the toxic effects of morphine and other of its derivatives.[1] Preliminary experimental studies with human beings showed that nalorphine reversed the respiratory depression caused by morphine, dihydromorphinone (Dilaudid) hydrochloride, methadone, meperidine, and the proprietary preparation containing alkaloids of opium, Pantopon.[2] Patients suffering from overdoses of methadone, racemorphan (Dromoran) hydrobromide, or dihydromorphinone have been successfully treated.[3] It seemed reasonable to believe, therefore, that nalorphine would be of benefit in treating heroin toxicity since heroin is a diacetyl derivative of morphine.

This report is concerned with the treatment of a patient suffering from acute heroin poisoning. To my knowledge, no instance of acute heroin toxicity treated with nalorphine has appeared in the literature.

REPORT OF A CASE

A 22-year-old married mulatto woman entered Kings County Hospital on May 1, 1953, at 10:35 p. m. in coma. Earlier that day, the patient had engaged in sexual contacts with several men who were heroin addicts. At about 6 p. m. the patient was given an unknown quantity of heroin intravenously by means of a hypodermic needle attached to a medicine dropper. The patient then apparently "blacked out" and could not recall any further drug administration. Later, the other addicts noted that the patient was breathing with difficulty and was cyanotic. They called the police and confessed that they had given her heroin. The police immediately brought the patient by ambulance to Kings County Hospital. This was allegedly the first experience the patient had with heroin, although she stated that she had smoked marihuana cigarettes and "sniffed" cocaine previously.

On admission, the patient was comatose, with respiratory arrest and pinpoint pupils. She was promptly placed in a respirator, but no improvement was noted. The patient appeared lifeless. She was removed from the respirator, and artificial respiration was administered manually. The patient was then given 1 cc. (5 mg.) of nalorphine intravenously. Within one minute, the patient started to breathe, her pupils dilated, and she rapidly became conscious and sat up. She then became belligerent.

Her personal and her family history as well as a system's review were noncontributory. The social history revealed that the patient was born in North Carolina and lived there most of her life. She was married and had two living children. She had left her husband seven months previously and had come to live in New York City. Life as a waitress was "dull," and the patient described herself as "depressed and unhappy." She seldom smoked and drank only small amounts of whisky.

Physical examination was performed after the patient had responded to nalorphine. The patient was a well-developed, well-nourished, light-skinned mulatto woman who was quarrelsome, confused, and somewhat somnolent. She vomited intermittently. The pertinent findings were: temperature, 98 F (by rectum); pulse rate, 100 per minute and thready; respirations, 16 per minute; blood pressure, 90/60 mm. Hg; and skin, warm and moist. No rashes or petechiae were noted. There was no external asymmetry of the head. The pupils were now dilated, round, and regular and responded well to light. Funduscopic examination was normal. The ear canals and drums were normal. There was no inflammation of the mouth. The neck was supple, and there was no venous distention. The thyroid gland was not palpable; the trachea was in the midline; and there were no palpable cervical nodes. The breasts and nipples were normal. There were no nodes or masses in either axilla. The respiratory excursions of the chest wall were slightly diminished bilaterally. There were moist, inspiratory and expiratory rales heard both anteriorly and posteriorly from the apexes to the bases. The heart sounds were somewhat obscured by the respiratory noises. No murmurs were audible. The apical rate was 100. The radial pulse was thready. The abdomen was soft, and the liver, spleen, kidneys, and masses were not felt. Pelvic examination showed that the cervix was firm and that an old laceration was present. There was slight tenderness on movement of the cervix. Rectal examination revealed no masses or tenderness. A needle puncture wound was noted in the right antecubital fossa. No lymph nodes were palpable. The cranial nerves were intact. Superficial and deep reflexes were normal. The gag reflex was present. Initial laboratory data included hemoglobin level 12 gm. per 100 cc. and white blood cell count 9,200 per cubic millimeter. The peripheral smear showed normocytic, normochromic red blood cells, adequate platelets, and no atypical cells. There were 75 polymorphs, 23 lymphocytes, and 2 monocytes per cubic millimeter. The urine was clear. Urinalysis showed no sugar, acetone, albumin, or bile. Microscopic examination of the sediment showed nothing abnormal.

While in the hospital, the patient received oxygen by mask, and her blood was typed and cross matched. A culture of the blood was made. She was then given 300,000 units of aqueous crystalline penicillin, 600,000 units of aqueous procaine penicillin, and 1 gm. of streptomycin, intramuscularly. An infusion of 2,000 cc. of isotonic sodium chloride solution containing 5% dextrose and water-soluble vitamins was given slowly during the first 24 hours. Intramuscular injections of 600,000 units of aqueous procaine penicillin once per day and 1 gm. of streptomycin twice per day were continued. The patient's blood pressure was low for the first 48 hours, with blood pressure readings varying from 70/40 to 100/52 mm. Hg. Intramuscular injections of 5 mg. of phenylephrine (Neo-Synephrine) hydrochloride were given whenever the systolic blood pressure dropped below 80 mm. Hg.

From the University Division Medical Service, the Kings County Hospital.

Drs. Martin Metz, Jacques Squillace, and Seymour Cutler assisted in the management of the patient.

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Twelve hours after admission, the temperature rose to 101.4 F. Another blood culture was made. A 12 lead electrocardiogram showed a sinus tachycardia at a rate of 100 per minute. The temperature remained at levels of 100.6 to 101.4 F for the first three days after admission and then became normal. About 24 hours after admission, a roentgenogram of the chest was taken (fig. 1). It was interpreted as showing bilateral, moderately extensive, symmetric, confluent areas of bronchopneumonic infiltration. The patient, clinically, had faint basal rales posteriorly at this time.

On the third day, the blood urea nitrogen was 24 mg. per 100 cc.; the fasting blood sugar level was 73 mg. per 100 cc.; and reaction to the Mazzini test was negative. The two blood cultures made previously showed no growth. The initial vomitus and urine specimens were reported as containing barbiturates (qualitative test). No attempt was made to identify heroin in the urine. On the fifth day after admission, the patient was afebrile, felt well, and was ambulatory. A repeat chest film (fig. 2) showed marked clearing of the bronchopneumonic infiltrations. Administration of the antibiotics was discontinued. The patient was discharged to the custody of the police department seven days after admission.

COMMENT

The use of nalorphine in this case was probably a life-saving measure in view of the poor initial response to treatment in the respirator. The finding of barbiturates in the vomitus and in the urine was not surprising as they are sometimes used to adulterate narcotics.[4]

It appeared that the presenting symptoms of coma, respiratory arrest, and pinpoint pupils were mainly caused by heroin. It is possible that part of the picture of coma, the prolonged hypotension and the lethargy,

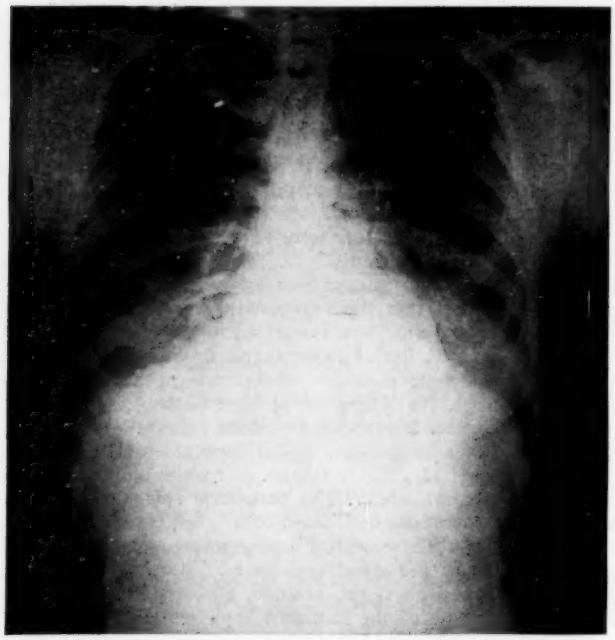

Fig. 1.—Roentgenogram showing bilateral, moderately extensive, symmetric, confluent areas of bronchopneumonic infiltration in patient with acute heroin intoxication.

may have been attributable to the barbiturates. Nalorphine probably did not reverse the effects of the barbiturates, as there are reports that it is ineffective in dealing with barbiturate toxicity.[2a] Dulfano, Mack, and Segal [5] however, have reported some success in combating barbiturate induced respiratory depression by using large (35 to 40 mg.) doses of nalorphine. Dulfano and coworkers [5] postulated that the mechanism of action of nalorphine was to stimulate the nervous system directly, particularly the respiratory center, and thereby to elevate the threshold of the center to depression by morphine. A competitive displacement between nalorphine and morphine in the enzyme-coenzyme systems affecting the central nervous system was offered as a possibility.

Nalorphine administered intravenously in 5 to 10 mg. doses to adults suffering from poisoning with morphine

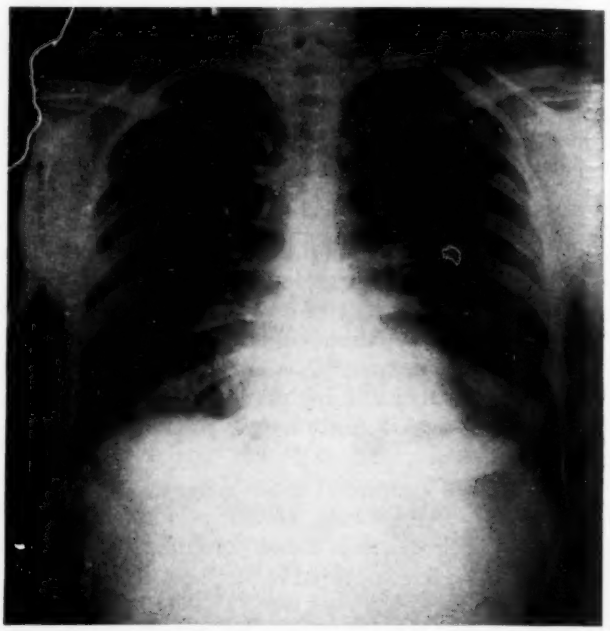

Fig. 2.—Roentgenogram showing marked clearing of bronchopneumonic infiltrations after administration of aqueous crystalline and aqueous procaine penicillin and streptomycin in same patient as in figure 1.

or its derivatives appears to be a therapeutic and safe dose. It may be repeated in 10 to 15 minutes, if adequate respiratory stimulation is not obtained. The effect lasts for 2 to 3 hours. It may be administered intramuscularly or subcutaneously, but, when rapid action is necessary, the intravenous route is preferred.

SUMMARY AND CONCLUSIONS

A case of acute heroin intoxication, with respiratory arrest treated initially with nalorphine (Nalline) hydrochloride, is reported. It appears that the toxic effects of heroin, as well as those of many other narcotics, can be reversed by administration of nalorphine.

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Rectal Examination in General Practice.—One of the most important maneuvers that the general practitioners should learn is to make a rectal examination with a minimum of discomfort to the patient, and at the same time gain a maximum amount of knowledge about the rectal condition. While proctologists feel that no rectal examination is complete without a sigmoidoscopic examination, followed, if indicated, by roentgenological studies, nevertheless, rectal examination, as it can be performed without special instruments, is often of great value and should not be omitted in a general physical examination.—S. B. Kleiner, M.D., Rectal Examination in General Practice, *Connecticut State Medical Journal*, August, 1953.

NERVOUS SYSTEM DAMAGE FOLLOWING PARAVERTEBRAL BLOCK WITH EFOCAINE

REPORT OF THREE CASES

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It has recently become well recognized that anesthetic agents injected into the subarachnoid space may damage nervous tissue [1] and cause meningeal irritation manifested by pleocytosis and increased protein in the spinal fluid.[2] Light and co-workers inferred that "probably there is in every case some toxic reaction produced in nervous tissue by the agents used for spinal anesthesia . . ." [3] Neurological sequelae of spinal anesthesia are sometimes severe and apparently permanent.[4] An almost unavoidable hazard of paravertebral nerve block is inadvertent injection of the agent used into the subarachnoid space.[5] This probably occurs because of an outward prolongation of the subarachnoid space or in other instances because of movement of the patient and tearing of the tissues after the needle is in place. At least one case of severe spinal cord damage following a paravertebral block has been reported.[6]

Efocaine is a relatively new anesthetic agent that offers the advantage of producing a much more prolonged local anesthesia than the usual agents.[7] It contains 1% procaine, 0.25% procaine hydrochloride, and 5% butyl-*p*-aminobenzoate in a solvent consisting of 2% polyethylene glycol-300, 78% propylene glycol, and water. Athough in preliminary animal experiments fibrous reaction was noted around nerve bundles after local injections of Efocaine,[8] it has been considered safe for use in paravertebral nerve blocks.[7] A recent report,[9] however, noted one instance of transverse myelitis and one of hemianhidrosis following similar blocks with Efocaine.

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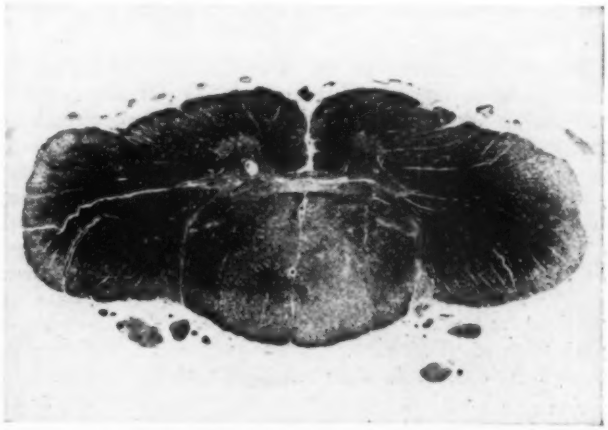
REPORT OF CASES

CASE 1.—A 63-year-old white man was given a paravertebral block with Efocaine from the fifth to the eighth dorsal segments on the left at the New York Hospital, on Jan. 10, 1953, because of chest pain associated with widespread inoperable thoracic carcinoma. There were no evidences of spinal metastases. Neurological examination on hospital admission revealed no defect. About two hours after the block the patient complained of inability to move his left leg. He soon noted weakness of his right leg and fecal incontinence, and a Foley catheter had to be used for urinary retention. When examined the next day, he was noted to have an incomplete transverse myelitis at about the fourth dorsal level. Thereafter he remained bedridden because of paraplegia, which improved only minimally.

Examination two and one-half months after the nerve block revealed severe weakness of the left leg, with increased resistance to passive motion and hyperactive deep tendon reflexes. On attempts to move the left leg, he reported "electrical shocks" through it. The left abdominal reflex was decreased. The right leg was also very weak. There was impairment of position sense in the toes of both feet and diminished perception of vibration from the iliac crest down on the left side and in the toes of the right foot. There was a band of decreased perception of pin and cotton between the fifth and eighth dorsal dermatomes on the left. Both plantar responses were equivocal. Sphincter function was intact, having been recovered about one and one-half months after the nerve block.



Loyez myelin sheath stain of the spinal cord at approximately the fourth dorsal level.

The patient died on April 18 after a gradually progressive downhill course characterized by deepening icterus. Autopsy revealed carcinoma of the pancreas, with widespread metastases but none affecting the cord. Examination of the spinal cord with Loyez myelin sheath stain demonstrated striking degeneration of the gracilis column and to a lesser extent of the peripheral layers of the cord (figure). The changes were present in the thoracic region and extended to the cervical level.

CASE 2.—A 25-year-old white woman with a 21 week intrauterine pregnancy, rheumatic heart disease with mitral stenosis, and an enlarged heart was subjected to a mitral commissurotomy at the New York Hospital on Feb. 18, 1953. The left auricle was noted to be free of thrombi. As a routine postoperative measure she received a paravertebral block with Efocaine from the second to the sixth dorsal level on the left (2 cc. for each nerve) before leaving the operating room. Two days later she complained of serious weakness of both legs.

Examination five days postoperatively revealed severe weakness of the left leg, slight weakness of the right leg, impaired recognition of pin and cotton from the level of the fourth dorsal dermatome caudad bilaterally (impairment being more

pronounced on the left), slight diminution of position sense in the toes of the left foot, and inability to void spontaneously.

She improved slowly and steadily thereafter, so that when last seen a month later sphincter function had returned and she could walk unaided with only slight difficulty. Examination at this time revealed slight weakness of the left leg, and hyperreflexia of both lower extremities. The hyperreflexia was more evident on the left, where there was a positive toe stretch response, unsustained ankle clonus, and an extensor plantar response. Rapid rhythmic alternating movements were impaired in the left leg; the left abdominal reflex was absent. There was diminished sensation to pin and cotton below the sixth dorsal level bilaterally that was slightly more pronounced on the left. Position and vibration sense were intact.

CASE 3.—A 40-year-old white man had the upper lobe of the right lung removed for pulmonary tuberculosis at the Castle Point Veterans Hospital on Jan. 2, 1953. At the conclusion of the operation the second through the ninth intercostal nerves on the right were each blocked with 2 cc. of Efocaine. Upon first regaining consciousness, the patient complained of inability to move his right leg. Within the next few days complete paralysis of this leg developed with a marked paresis of the left lower extremity and loss of anal, rectal, and bladder control and sensation. About one week after the operation he began to improve, and had continued to improve very slowly to the time of writing. Three lumbar punctures revealed no remarkable findings save for protein levels of 50, 90, and 52 mg. per 100 cc.

He was transferred to the Bronx Veterans Hospital for neurological opinion. Here, examination about one and one-half months after the accident revealed absent abdominal and cremasteric reflexes, weakness of the right abdominal musculature, almost complete paralysis of the right lower extremity with increased resistance to passive motion, and slight weakness of the left lower extremity. Knee and ankle jerks were hyperactive bilaterally: there was an extensor plantar response bilaterally that was more easily elicited on the right. Sustained patellar and ankle clonus was present on the right, with unsustained ankle clonus on the left. There was decreased recognition of pin, cotton, and temperature stimuli from the nipple line down on the right and from the costal margin down on the left. This loss was more conspicuous on the right side. There was severe decrease of position and vibration sense in the right lower extremity and slight diminution of vibration in the left leg. During the ensuing month the patient regained anal and rectal sensation and control. When transferred back to the Castle Point Veterans Hospital two and one-half months after operation, he was just beginning to become aware of bladder fulness. Final diagnosis was myelopathy secondary to Efocaine.

COMMENT

These three patients were called to our attention within a period of less than two months. In two of them the "blocks" were accomplished by fully trained anesthesiologists. It seems highly likely that in each case some Efocaine permeated the subarachnoid space and was there absorbed to cause local myelopathy.

SUMMARY AND CONCLUSIONS

Three patients with evidence of spinal cord damage following paravertebral thoracic nerve block with Efocaine are reported on. In one, postmortem findings demonstrated extensive damage to the periphery of the cord and dorsal columns. Paravertebral nerve block carries dangers of damage to nervous tissue that are related to those of spinal anesthesia but not so great. Efocaine is not a safe agent for use in paravertebral nerve blocks.

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PHRENICECTOMY AS AID TO REPAIR OF LARGE ABDOMINAL HERNIAS

Arthur S. W. Touroff, M.D., New York

During the repair of certain hernias, considerable difficulty may be encountered in replacing the contents of the sac into the abdomen. This is especially apt to be the case when the hernia is very large and of long duration for, under such circumstances, the abdominal cavity may be too small to readily accommodate the viscera that have been displaced from it. In the case of interstitial, inguino-scrotal-abdominal hernia about to be described, the aforementioned difficulty was overcome by performing a phrenicectomy several days prior to hernioplasty. Inasmuch as I was unable to find any report of similar surgical management of a large inguinal hernia, the case appears worthy of recording.

REPORT OF A CASE

A 60-year-old man first consulted me on May 13, 1953, because of a large, slowly developing right-sided abdominal mass of 30 years' duration. The diagnosis of congenital right inguinal hernia had been made shortly after birth. The mass remained essentially unchanged in size for approximately 30 years. Soon thereafter, it began to enlarge slowly but progressively. It first descended into the scrotum but, at the end of five years, began to extend in an upward direction over Poupart's ligament. Thereafter, it enlarged steadily upward and laterally upon the abdominal wall to form a prominent, somewhat irregular superficial mass that covered the right lower and lateral abdominal areas and the right thigh.

As the mass became larger, the patient began to experience "dragging" pain and "heaviness" in the lower abdomen. During the last 10 years he experienced increasing discomfort while sitting, especially while at work as a sewing machine operator. As the abdominal component of the mass increased in size, it became increasingly noticeable beneath his clothing, which had to be let out progressively. He did not consult a physician at any time for fear that operation would be advised, but finally sought medical advice because the abdominal discomfort handicapped him in his work.

His medical history included removal of a cataract from the right eye six years previously and recurrence of seasonal hay fever for the last 35 years. On physical examination, the patient was well developed and nourished. The relevant physical findings were confined to the local area where a large, irregular, superficial mass covered the right lower and lateral abdominal wall and the right thigh. The uppermost portion of the mass was situated well out in the right flank, at a level 2 in. (5.08 cm.) above that of the umbilicus. From there, it extended downward and mesially for a distance of 16 in. (40.64 cm.) to the bottom of the distended scrotum. It measured a little more than 8 in. (20.32 cm.) in its widest transverse diameter and protruded about 8 in. above the level of the surrounding abdominal wall (fig. 1). The mass was covered by thin, tightly stretched skin and was fluctuant in various areas.

The bulk of the lesion was slightly movable, but the lower portion was somewhat fixed in the inguinal region. In the latter area, the deep surface of the mass could be traced to a widely dilated, external ring that measured about 3½ in. (8.89 cm.) in diameter. The right testis could not be felt. The impression gained was that the lesion was a congenital inguinal hernia that had descended into the scrotum and then turned upward, superficial to Poupart's ligament, to spread out over the abdominal wall beneath the skin and subcutaneous tissues.

In view of the increasing abdominal discomfort and interference with ability to earn a livelihood, the patient was advised to undergo surgery. He entered Mount Sinai Hospital

Surgeon to the Mount Sinai Hospital.

on May 20, 1953. A gastrointestinal series and barium enema performed shortly after admission revealed the small bowel distal to the proximal jejunum and the caput coli, ascending colon, and proximal half of the transverse colon within the hernial sac. The stomach was stretched, the distal half being drawn more beyond the midline than normal. Examination of the chest revealed no abnormalities (fig. 2). On May 25, as a preliminary to hernioplasty, left phrenicectomy was performed with the man under local anesthesia. Within the next two days, the abdominal mass became perceptibly smaller and less tense.

Fig. 1.—Preoperative appearance of hernial mass that has spread upward and laterally from inguinal region to overlie the entire right lower and lateral abdominal wall and the right thigh.

On May 27, right hernioplasty and orchidectomy were performed with combined thiopental (Pentothal) sodium and spinal anesthesia. A 10 in. (25.4 cm.) right inguinal incision was made over the mass and the sac exposed and dissected away from the surrounding tissues. The sac was opened and a large amount of thin, straw-colored fluid aspirated. After the latter had been accomplished, the sac was opened widely. It contained a large mass of thickened, hypertrophic large bowel and indurated, congested mesocolon and omentum that were firmly adherent to the interior of the sac. An extensive collateral network of distended blood vessels extended between the adherent bowel and omentum and the interior of the sac. The right testis was intimately adherent to the outer aspect of the sac close to the uppermost portion of the latter. From there, the thickened spermatic cord pursued a tortuous downward and mesial course, on the sac, to the bottom of the scrotum. It then turned upward and passed through the external inguinal ring, mesial to the neck of the sac. The inguinal ring was approximately 3½ in. in diameter; its margins were firm and well defined.

The inguinal canal was opened in the usual manner and the neck of the sac isolated. The contents of the sac were freed, and as much of the indurated omentum as possible was excised. The bowel and mesocolon then were replaced in the abdomen with ease, and the sac was excised as were the testis and tortuous spermatic cord. (Orchidectomy was done to insure complete closure of the hernial defect.) The neck of the sac was closed with continuous silk sutures. The transversalis fascia was plicated with interrupted sutures and a typical Andrews-Bassini repair performed. The large mass of redundant skin and subcutaneous tissue that formerly covered the hernial mass was excised, and the edges of the wound were united with interrupted silk sutures. A cigarette drain was placed beneath the suture line and brought out through the upper angle of the wound. A similar drain was brought out through a stab wound made in the most dependent portion of the right scrotal sac.

The postoperative course was relatively uneventful, except for the development of partial atalectasis of the left lower lobe following the phrenicectomy. The patient was permitted out of bed on the second day after operation. The dead space beneath the suture line obliterated slowly, and, because of a tendency for fluid to collect, the drains were kept in place for more than three weeks. At the time of the patient's discharge from the hospital on June 26, the wound was firmly healed (fig. 3). Since then, the patient has remained entirely free of symptoms.

COMMENT

The normally yielding walls of the abdominal cavity permit the capacity of that structure to vary in accordance with changes in the volume of its contents. Thus, in the presence of marked ascites or a massive intra-abdominal tumor, the peritoneal cavity is capable of substantial enlargement. Conversely, the peritoneal cavity may become significantly smaller after a large mass of intra-abdominal viscera have escaped through a hernial defect, especially if the latter is of long standing. Under such circumstances, an attempt at sudden replacement of the herniated visceral mass into the shrunken abdominal cavity, during the course of hernial repair, may be exceedingly difficult or even impossible. Such a problem

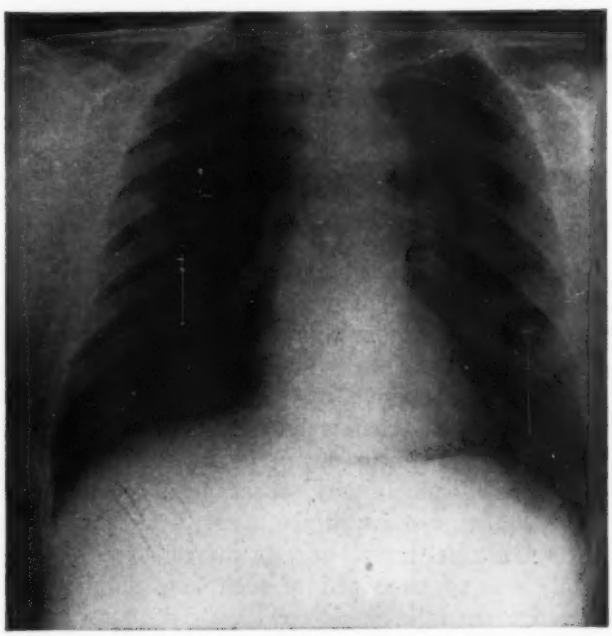

Fig. 2.—Preoperative film of chest showing left side of diaphragm at level of 10th rib, laterally.

is encountered not infrequently in attempting to repair a massive omphalocele or a large congenital diaphragmatic hernia in an adult. In the former instance, the operation sometimes is performed in two stages. The first stage consists merely of closing the skin over the visceral protrusion. After the peritoneal cavity enlarges sufficiently to accomodate the replaced mass of viscera,

the second stage, consisting of repair of the muscular and fascial defect in the anterior abdominal wall, is carried out. In the case of repair of a large diaphragmatic hernia of long standing, phrenicectomy is often performed in order to enlarge the peritoneal cavity. This may be done either at the time of diaphragmatic repair or as an independent preliminary procedure.

Fig. 3.—Four weeks after operation there is long operative scar and absence of previous abdominal and right scrotal masses.

Of all the structures that constitute the boundaries of the abdominal cavity, the diaphragm is the thinnest and also the most yielding. Furthermore, unlike any other large muscle, its tonus depends almost exclusively upon the integrity of a single nerve, namely the phrenic nerve. Interruption of the latter, either temporarily or permanently, results in prompt paralysis and elevation of the diaphragm, which in turn immediately increases the capacity of the abdominal cavity. Thus, phrenic interruption constitutes an extremely effective method of facilitating operative repair of any large abdominal hernia when the abdominal cavity has shrunken as the result of long-standing, massive displacement of viscera into an extra-abdominal hernial sac. In the case reported, the hernia was so large and of such long duration that operative repair in one stage appeared almost impossible. Accordingly, an attempt to enlarge the abdominal cavity by a left phrenicectomy appeared worthy of trial.

The efficacy of the procedure was quickly demonstrated by a noticeable reduction in the size and tenseness of the hernial mass. Graphic evidence of enlargement of the peritoneal cavity was subsequently afforded by comparing the chest films taken before and after phrenicectomy. The preoperative film revealed the left diaphragm situated at the level of the 10th rib laterally, while the postoperative film showed that it had risen to the level of the 6th rib (figures 2 and 4). Additional evidence of the effectiveness of the phrenic interruption was obtained by comparing the original abdominal roentgen findings with the findings at the time of hernioplasty. The former disclosed a great mass of large and small bowel within the hernial sac; the latter revealed only a long loop of proximal colon together with omentum and fluid within the sac. (This indicated that the abdominal cavity had enlarged sufficiently after the phrenicectomy to permit spontaneous reduction of a large part of the contents of the hernial sac.) Additional evidence of significant enlargement of the peritoneal cavity as a result of phrenicectomy consisted of the ease with which the bowel could be returned to the abdomen during the hernioplasty. Finally roentgen examination performed about a week prior to the patient's release from the hospital revealed the barium-filled splenic flexure of the colon to be lying at an unusually high level beneath the markedly elevated, paralyzed left side of the diaphragm.

SUMMARY AND CONCLUSIONS

A case is presented in which an interstitial, congenital, right inguinal hernia enlarged to such proportions that it covered the entire right lower and lateral abdominal wall and the upper portion of the right thigh. The mass was of such large size and long duration that the abdominal cavity appeared too small to accomodate it, if hernioplasty were performed in one stage. Radical cure was achieved by performing phrenicectomy in order to increase the capicity of the peritoneal cavity followed, two days later, by an Andrews-Bassini hernioplasty and orchidectomy. The method appears worthy of further trial in all cases of abdominal hernia of large size and long duration.

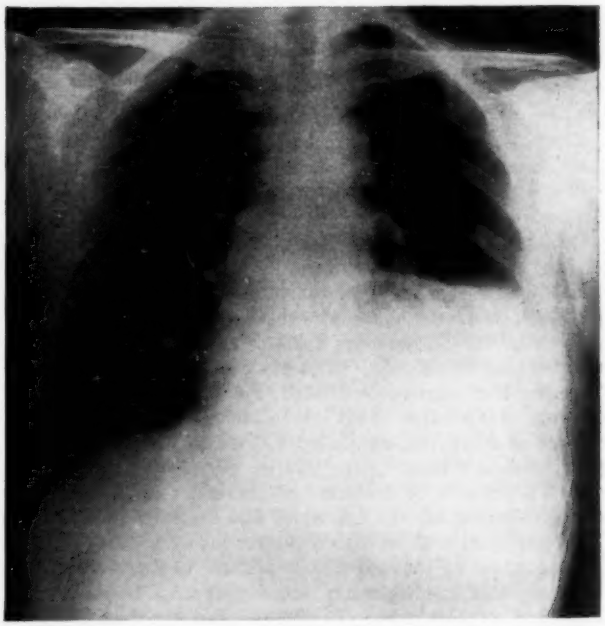

Fig. 4.—Postoperative film of chest showing paralyzed left side of diaphragm at level of sixth rib, laterally, after phrenicectomy.

994 Fifth Ave. (28).

GENITAL WARTS—A VENEREAL DISEASE

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Lieut. John D. Silbar
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The number of cases of genital warts (condyloma accuminata, verruca accuminata) has been noted to increase tremendously on the establishment of a replacement center at Camp Atterbury, Indiana. After treating approximately 50 cases of these lesions and seeing the high incidence in the troops returning from overseas compared to civilians or soldiers from the United States, we have felt that a closer evaluation should be made. After a study of 70 men and 24 women, we are making a preliminary report because we feel the medical profession should be alerted to this problem for the reasons to be discussed.

TREATMENT IN MEN

Complete studies of 70 men have been carried out (fig. 1). Of these patients, 65 had been stationed in Korea and Japan, 4 had never been out of the United States, and one was in Okinawa. All 65 patients who were in the Far East admitted contact with native women. Other venereal diseases found were syphilis in one case; gonorrhea in 12, and penile ulcerations, type unknown, in 12. Only one of these 70 men was circumcised at birth, and the type of wart was smaller and more keratotic in this patient. These patients were treated by four methods: (1) application of 20% podophyllin in tincture of benzoin; (2) application of 20% podophyllin in tincture of benzoin, followed by circumcision; (3) circumcision alone; and (4) fulguration followed by circumcision.

A total of 57 patients were treated by application of 20% podophyllin in tincture of benzoin. We have used the following technique: 20% podophyllin in tincture of benzoin was applied directly to the lesions by sterile applicator. This was allowed to dry before the foreskin was pulled forward (three to four minutes); the patient was then instructed to wash the medication off completely in exactly two hours with soap and water. Reactions from this treatment were seen in only three cases; one was due to neglect in washing, and one patient was not able to retract his foreskin to cleanse the area. A total of 28 patients were seen here with recurrences following the use of podophyllin at other Army installations. Recurrences were re-treated by this technique at this hospital one to three times, with subsequent complete disappearance of the warts. These patients are now undergoing a long term follow-up, which will be reported in the future.

Thirteen patients were treated by use of 20% podophyllin in tincture of benzoin, followed by circumcision. There were no recurrences in this group. Four patients were treated by circumcision alone. Two of these had complete disappearance of the warts, and two required podophyllin treatment after circumcision for complete eradication of the disease. Five patients were treated by fulguration with electric cautery, with complete disappearance. One patient required two treatments by this method. It is of interest that in one patient with multiple warts they disappeared spontaneously.

From the Sections of Obstetrics and Gynecology, Urology, and Dermatology, U. S. Army Hospital, Camp Atterbury, Indiana.

TREATMENT IN WOMEN

At the time of writing 24 women were under treatment for genital warts (fig. 2). They were all wives of returnees from the Far East. All of the husbands admitted having had sexual relations with native women while in Japan or Korea, and 22 of these men were under treatment for penile lesions. The remaining two men admitted having had warts while overseas. Nine of the women were pregnant, and two were within four to six weeks post partum.

The majority of these women noticed the appearance of the lesions four to six weeks after their husbands returned from the Far East. Vaginal and urethral smears have been consistently negative for the presence of gonococcic organisms, and results of serologic studies in all cases were negative.

Fig. 1.—A typical lesion found in men with genital warts before treatment.

These lesions were treated by local application of podophyllin in tincture of benzoin to each individual wart. If an unsatisfactory response was obtained with this regimen, then excision of the lesions either by electrodissection or by sharp dissection was done.

COMMENT

We have attempted to show the etiological relationship between sexual intercourse and genital warts. All of the consorts of the women studied had genital warts prior to the appearance of similar lesions in the women. Our data show that 66 out of 70 patients had intercourse in the Far East, and the incidence of genital warts was in sharp contrast to the rarity of genital warts among troops who have never been to the Far East. The 100% incidence of genital lesions in the women in whose consorts

warts developed following service in the Far East is to us proof of the infectious nature of the disease and its mode of transmission.

SUMMARY

The incidence of genital warts at Camp Atterbury, Indiana, has been exceedingly high. The data reported point to sexual intercourse as the mode of transmission of the virus producing genital warts. This seemingly justifies the classification of genital warts (condyloma accuminata, verruca accuminata) as a venereal disease.

Fig. 2.—A typical lesion found in women with genital warts before treatment.

The Cause of Hypertension.—There are times when the strident conflicts of investigators, the rise and fall of theories and the babel of unsorted evidence create such a sense of confusion in a field that an atmosphere of frustration and despair prevails in which no one believes anything; this has happened to some extent in hypertension. Yet somewhere between the extreme skepticism both of the very young and of the old and tired, and the transient enthusiasms of the protagonists of this theory or that remedy, lies a middle ground of achievement and even agreement. It is generally agreed, for example, that the cause of ordinary hypertension is narrowing of the terminal vascular bed of the systemic circulation, especially the arterioles. It is also generally believed, although with some dissension, that there are three major causes of such narrowing. One is increased discharge via the sympathetic nervous system. Another is increased vasomotor tone produced either by circulating substances or by metabolic changes. A final cause is structural changes in the vessels themselves, frequently influenced at least in part by the first two factors. . . . In the ordinary hypertensive the neurogenic factor is usually the first and, as believed by some, the initiating factor in the process. The humoral or metabolic element contributes later, when the kidney participates in the disease. This factor accelerates the disease by increasing vasomotor tone, and, finally, the structural changes in the arterioles produced in part by these influences turn the screw tighter.—Milton Mendlowitz, M.D., The Treatment of Hypertension with Drugs, *Annals of Internal Medicine,* November, 1953.

EXTENSION TRACTION WITH PERFORATED FOAM RUBBER STRIPS

Maurice Gershman, M.D., Far Rockaway, N. Y.

The application of perforated foam rubber strips with rayon or drill cloth backing to an extremity is a type of extension traction that I have found to be simple and easy to apply. Foam rubber consists of an infinite number of air bubbles in a matrix of rubber. As this mass is sliced longitudinally, there is created a gripping surface consisting of many concavities with rubber septums. Perforations made in the rubber permit an even better grip on the surface and allow air to reach the skin. The septums act as treads against the skin and hair. Hair acts advantageously when foam rubber is used, in contrast to the discomfort it causes when adhesive plaster is used. The foam rubber is held snugly and smoothly against the skin by an elastic bandage. No shaving or other preparation of the skin is required.

This method is particularly useful in applying traction to the limbs of elderly patients who have thin atrophic skin of poor quality, and it is excellent for use after hip surgery when traction is to be maintained only until a brace can be applied. Its ease of removal and reapplication make it particularly useful while measurements and fittings of a brace are being carried out. When bilateral traction is being used in the conservative management of the acute herniated disk these foam rubber strips can be readily removed and reapplied to facilitate examination of the patient. This would allow bathroom privileges if the traction did not have to be applied continuously. Its use as traction in the guillotine amputation is recommended. Up to 10 lb. (4.5 kg.) of traction has been used for short periods without slipping.

A spreader, consisting of a square wooden block, is used as in other forms of skin traction, to prevent compression of the skin over the bony prominences. The directions for application of this type of traction are as follows. 1. Place the eye hook of the spreader through a slit made in the center of the perforated foam rubber strip. 2. Use the thumb tacks to hold the strip smoothly against the covered surface and edges of the wooden spreader, and secure the tacks by encircling them and the spreader with ½ in. adhesive tape. 3. Place the spreader one inch beyond the extended limb, and hold the two free ends of the strip snugly and smoothly with the foam rubber against the skin. 4. Start the elasticized bandage at the two free ends of the strip, encircling the limb to

Fig. 1.—Application of traction to lower extremity.

hold the strip securely. 5. Tie the rope to the eye hook of the spreader, and then attach the proper weights, through a pulley if possible, for traction.

Up to the present moleskin has been advised as the best material for skin traction, but I believe that foam rubber has the following advantages over moleskin. 1. Moleskin tends to deteriorate in warm weather. 2. Some type of adherent substance must be painted on the skin when using moleskin. 3. Glue tends to harden, and frequently excoriation and blistering occurs. 4. Preparation of the skin consisting of shaving followed by thorough cleansing with alcohol and ether is needed before glue is applied. This procedure is time consuming and requires trained personnel to be in attendance. 5. Foam rubber can be sterilized in an autoclave for use over compounded areas, and it can be washed for reuse.

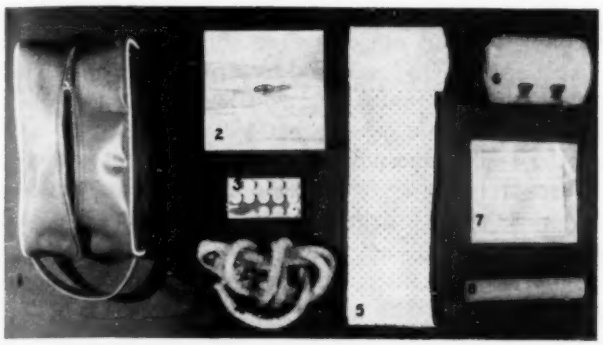

Fig. 2.—1. Trac-Kit (holding all contents). 2. Spreader with eye hook. 3. Thumb tacks. 4. Three feet of clothes line cord. 5. Perforated foam rubber strip with drill cloth backing. 6. Three inch elasticized bandage. 7. Sterile gauze pad. 8. Four inch dowel for hand grip.

We first used foam rubber that was adherent to the regular broadloom carpeting, but now foam rubber of ⅛ in. thickness has become a universal material and can be attached to almost any backing material that can be perforated. It has a wide field of application in the realm of public safety during national emergencies, as it can easily be placed in a first aid kit (fig. 2) and applied by inexperienced persons, if necessary.

1210 Cornaga Ave.

Female Surgical Patients.—One of the largest groups of neglected patients is made up of women who have been operated on. A surgeon usually makes rounds rather hurriedly and discharges his patient while he is on his feet. That is poor technique. Most women have a lot of worries that have been bothering them and cannot bring themselves to ask questions of a man in a hurry. That can be easily corrected. A couple of days before contemplated discharge from the hospital, tell the patient to get a big piece of paper and a pencil, and put down and number all the questions regarding her health that have disturbed her. . . . When the surgeon is ready to send the patient home, he should sit down with the list in hand and discuss each of the items enumerated. They can usually be disposed of to the satisfaction of the patient. He should give specific instructions regarding domestic work, sex life, and driving automobiles. Cancerphobia must be firmly removed. A surgeon's job, however skillful his operating, is not complete until he has set his patient's mind at rest.—R. T. Richards, M.D., Surgeons and Neuroses, *The American Surgeon,* September, 1953.

GOUT IN A NEGRO WOMAN

REPORT OF A CASE

Harry Bartfeld, M.D., New York

Gout is a disease rarely seen in women; about 98% of all cases are in men. It is said to occur infrequently in Negroes, but cases appear especially in clinics where a large number of Negroes are in attendance. It was never reported as appearing in a Negro woman until 1953.[1] This case meets the criteria for gout particularly for clinical signs, hyperuricemia, and identification of uric acid tophi. The case being presented here is that of a Negro woman who has had gout over a period of six years.

REPORT OF A CASE

On May 27, 1952, at the age of 60 a Negro woman was admitted to Bellevue Hospital complaining of painful swelling of the left ankle and left great toe for two weeks and painful swelling of the right ankle and right great toe for four days. She added that at one time white cheesy material ("worms") was discharged from the left toe. In addition, she complained of dizziness and dyspnea. This was her second attack of acute gout; the first occurred five years before beginning in her right ankle and then involving her right great toe; it was so severe that she could not walk. Two years before admission, she was discovered to be hypertensive, and symptoms of dyspnea and orthopnea developed for which she received digitalis and meralluride (Mercuhydrin) sodium. In 1936, she was admitted to Bellevue Hospital for right shoulder and back pain. A roentgenogram taken then showed osteoarthritic changes of the lumbar vertebrae. At that time, she was drinking to excess. There was no history of venereal disease. Her menses stopped in 1928. Her family history indicated that neither her father nor her mother had gout. Of her five brothers and two children, none ever had gout.

Physical examination revealed a well-nourished woman who appeared chronically ill. The head had no abnormalities, except for healed scars of an old burn over the nose and cheeks. One breast had keloid scars. The lungs were normal, except for decreased breath sounds and decreased resonance of right base. The heart was enlarged to the left on percussion; the pulse rate and the ventricular rate were 80; and the blood pressure was 104/84 mm. Hg. The abdomen was soft; the liver edge was palpable just below the costal margin; and no masses were palpated. The limbs showed no cyanosis, and there was no clubbing of the extremities. The ankles were tender, warm, and swollen, and motion was painful. The metatarsophalangeal joints of both great toes were swollen, tender, and painful on motion. There was moderate bilateral hallux valgus. Roentgenograms of the hands, feet, and ankles showed generalized osteoporosis. In addition, roentgenograms of the feet showed changes typical of gout, namely, cresentic irregular destructive erosions between the first cuneiform and first metatarsal bones together with destructive areas in the heads of the first metatarsal and other metatarsal bones. There were also severe productive changes (figure).

Laboratory examination showed a moderate anemia; white blood cells 6,700 per cubic millimeter; sedimentation rate 74 mm. per hour; and hematocrit 36. The urine was normal except for albumin, 1+. The blood sugar was 80 mg. per 100 cc.; blood urea nitrogen 13 mg. per 100 cc.; and uric acid

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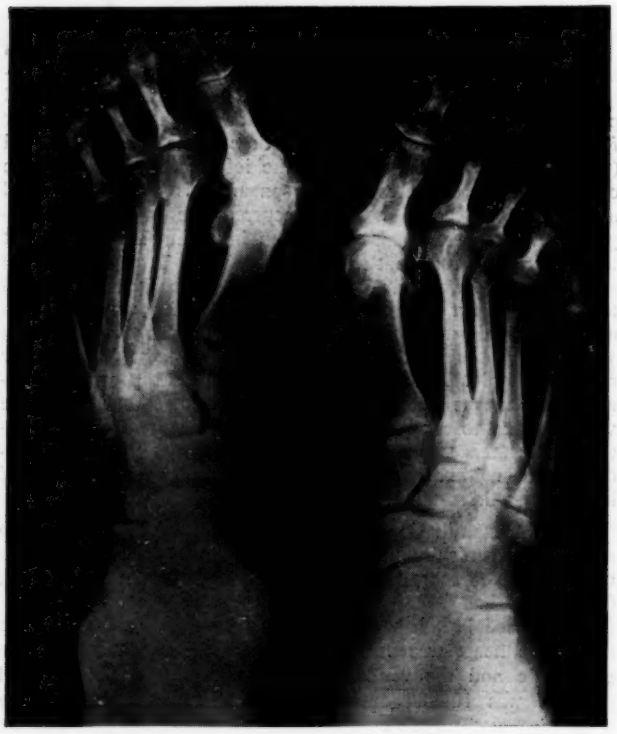
Dr. Edward F. Hartung helped in the preparation of the manuscript.

1. Perlman, L.; Bernstein, A.; Maslow, W. C., and Scatliff, J. H.: Gout in a Negro Woman, J. A. M. A. **151**: 726-728 (Feb. 28) 1953.

in whole blood 7 mg. per 100 cc. The uric acid value dropped to 5.1 mg. per 100 cc. The electrocardiogram showed some effect of digitalis.

In view of the low blood pressure and a history of hypertension, a diagnosis of Addison's disease was entertained, but laboratory studies did not verify this. The arthritis improved with rest and aspirin. As the swelling of the woman's great toes decreased, cyanosis and desquamation of the skin occurred. Her blood pressure rose to 144/110 mm. Hg, and she was discharged on June 19, 1952, with the diagnoses of gouty arthritis and hypertensive cardiovascular disease.

The patient was followed in the arthritis clinic of Bellevue Hospital for a period of six months after her discharge. During this time she had symptoms of chronic gouty arthritis, and at times her great toes were swollen and tender. Aspirin gave only moderate relief. A course of phenylbutazone (Butazolidine) was given but had to be discontinued as the drug not only made her nauseated but had no beneficial effect. She was treated with colchiceine (a derivative of colchicine) for



Roentgenogram of feet showing cresentic irregular destructive erosions between first cuneiform and first metatarsal bones and also destructive areas in heads of metatarsal bones.

four weeks, receiving oral doses as high as 1/25 grain (2.5 mg.) five times a day. This drug had no effect on the symptoms and caused no untoward effects. However, doses of 1/25 grain given six times a day had to be discontinued because diarrhea developed after three days.

On Dec. 5, 1952, the woman was admitted to Bellevue Hospital for symptoms of dysphagia associated with a 25 lb. (11.3 kg.) weight loss over a period of two months. On admission her symptoms of gouty arthritis were minimal, and the uric acid level for whole blood was 2.4 mg. per 100 cc. Esophagram studies revealed extrinsic pressure from an enlarged aorta. On the eighth hospital day, her temperature suddenly rose to 102 F, and her right ankle joint became red, warm, swollen, and extremely tender to slight touch and motion. A uric acid level in whole blood at this time was 6.8 mg. She was immediately given 1/100 grain (0.6 mg.) of colchicine hourly for six doses. Relief was dramatic, and the temperature was normal by the next day. A maintenance dose of 0.6 mg. of colchicine three times a day was continued until she was discharged improved on Dec. 23, 1952.

The woman was again admitted to Bellevue Hospital on Feb. 5, 1953, suffering with pain, swelling, and redness of both her ankles for six days and fever of 102 F on the day of admission. Laboratory studies revealed uric acid in whole blood 8 mg. per 100 cc.; cephalin-cholesterol flocculation test negative; serum total proteins 6.3 gm.; albumin 3.4 gm.; globulin 2.9 gm. per 100 cc.; alkaline phosphatase 3.5 units; and serum calcium 9.9 mg. per 100 cc. The sedimentation rate was 58 mm. in one hour. A complete blood cell count showed moderate anemia and 6,450 leukocytes per cubic millimeter, with 61 neutrophils, 27 lymphocytes, and 12 monocytes. Reaction to a Mazzini test was negative. The urine was normal.

On admission administration of colchicine, 0.6 mg. every hour was begun. Diarrhea developed after eight doses, and the symptoms subsided. A similar course of colchicine was given the next day, and the woman was then given a maintenance dose of 0.6 mg. of colchicine once a day until her discharge on Feb. 12, 1953.

COMMENT

This case meets the criteria of the American Rheumatism Association for gouty arthritis, namely a history of the first attack in a great toe; suddenness of an acute attack after some type of trauma (in this case after esophogram and other studies done because of dysphagia and weight loss); and hyperuricemia, aptly illustrated when an initial uric acid level for whole blood of 2.4 mg. rose to 6.8 mg. per 100 cc. during an acute attack. Tophi were never seen in this patient, although she reports that at one time a white cheesy material ("worms") was discharged from the side of the left great toe. Roentgenologic changes in the feet typical of gout are present in addition to productive changes often associated with a long-standing case of gout. The history of an initial attack five years before with none between and terminating in a chronic state with exacerbative episodes at more frequent intervals and involving other joints is also indicative of gout.

SUMMARY

A case of gouty arthritis in a Negro woman is presented. This is the second case to be reported.

135 W. 65th St. (21).

DIFFUSE INTERSTITIAL FIBROSING PNEUMONITIS

J. J. Kirshner, M.D.
R. L. Breckenridge, M.D.
F. A. Allbritten Jr., M.D.
and
P. A. Theodos, M.D., Philadelphia

Since Hamman and Rich in 1944 reported on four patients whose lungs, on autopsy, revealed diffuse interstitial fibrosis, a number of additional reports have appeared giving details of the clinical and pathological aspects of this disease. The course of the disease is progressively downhill, and death results from either respiratory failure or cardiac decompensation. In each case the diagnosis is made from study of autopsy material.

The following case report is considered of interest not only because it is another instance of this disease but also

From the Department of Medicine, Barton Memorial Division (Drs. Kirshner and Theodos), the Department of Pathology (Dr. Breckenridge), and the Department of Surgery (Dr. Allbritten), Jefferson Medical College.

because, in this instance, diagnosis was made by lung biopsy during the course of the disease and later confirmed by postmortem study.

REPORT OF A CASE

A 57-year-old man, a plate grainer by occupation, was admitted to Barton Memorial Division of Jefferson Hospital on Aug. 18, 1952, complaining of cough, dyspnea, and swelling of the ankles. His history included frequent nose bleeds since the age of 10 and growing pains at 13. The onset of exertional dyspnea occurred about three years prior to admission, but apparently did not merit medical attention. In November, 1951, while at work, he had an episode of dull retrosternal pain associated with moderate dyspnea. He was told by his family physician that he had heart failure, and he was given digitalis and mercurials. He remained fairly well until July, 1952, when the marked dyspnea and ankle swelling recurred. He stated that he first noted the blueness of his fingernails and enlargement of the ends of his fingers and toes at this time. He did not respond to treatment very well and was referred to the hospital for admission. His family history and a systemic review were noncontributory. He smoked 30 cigarettes daily and drank a fifth of a gallon of whisky a week.

Physical examination on admission revealed a thin white man who was orthopneic and markedly cyanotic. There were many prominent venules on the malar eminences and nose, a few spider nevi on the anterior thoracic wall, and distended subcutaneous veins on the chest wall. The cervical and peripheral veins were distended. There was marked kyphosis of the dorsal spine, with a great increase in the anteroposterior diameter of the thoracic cage. Respiratory excursions were equal but greatly diminished, with intercostal retraction on inspiration. There was slightly increased tactile fremitus at both bases with hyperresonance to percussion throughout. Breath sounds were diminished throughout both lung fields, and there were crackling rales in both lung bases. The apex beat was in the seventh intercostal space just medial to the midclavicular line; there were no palpable thrills; there was normal sinus rate and rhythm; and the first sound in the mitral area was accentuated and occasionally reduplicated. There was a grade 1 apical systolic murmur, and the second sound in the pulmonic area was soft. The liver was palpable three fingerbreadths below the right costal margin. There was no splenomegaly. The peripheral pulses were normal. There was a 2+ pitting edema of the ankles. Results of neurological examination were normal. Fundoscopy revealed a grade 1 retinal arteriosclerosis. Blood pressure on the right arm was 105/65, left arm 110/65, and right leg 120/80 mm. Hg. There was marked clubbing of the fingers and toes.

Initial laboratory studies revealed a hemoglobin level of 17.5 gm. per 100 cc., or 17%; red blood cells, 6,700,000 per cubic millimeter; white blood cells, 8,500; and hematocrit, 65%. Urinalysis was normal as were the results of Wassermann and Kahn tests. There were 6.36 gm. of total plasma proteins per 100 cc., with 3.86 gm. of albumin and 2.5% globulin. The sedimentation rate was 2 mm. in one hour (uncorrected); thymol turbidity was 1.8 units; and cephalin-cholesterol flocculation was 3+. The serum phosphorus was 4.1 mg. per 100 cc. and alkaline phosphatase 8.21 mg. per 100 cc. Cultures of four sputums did not yield acid-fast bacteria. The stool did not contain occult blood. The blood urea nitrogen value was 10.6 mg. per 100 cc.

An initial chest film revealed vascular congestion, pulmonary emphysema and fibrosis, calcification of the aorta, and prominence of the pulmonary artery segment of the heart (fig. 1). An electrocardiogram revealed right axis deviation, inversion of the T-waves in leads 2, 3, and AVF, and flattening in most of the chest leads. The patient was treated by bed rest, ammonium chloride, mucurial diuretics and digitalis, venesections, and oxygen. There was subjective and clinical improvement. At this time the patient's venous pressure and circulation times (both arm to lung and arm to tongue) were normal. Serum electrolytes and carbon dioxide combining power were normal.

It was felt at this time that the patient might have Ayerza's syndrome (syphilis of the pulmonary artery), with secondary polycythemia and cor pulmonale. For that reason, a lung biopsy was performed on Sept. 5, 1952. The microscopic section revealed chronic interstitial fibrosing pneumonitis (fig. 2). Repeated venesections were performed until the patient's hematocrit was 45%; it was maintained at that value. On Oct. 9, 1952, thrombophlebitis of the left calf developed. Anticoagulant therapy, penicillin, and bed rest were instituted until the symptoms and temperature had been normal for one week.

The patient was discharged on Oct. 28, 1952, feeling subjectively better, but was still dyspneic and cyanotic. He was seen by the hematology department and was given 5 mcg. of radioactive phosphorus for polycythemia. He apparently remained well for two weeks; then dyspnea, cough, ankle edema, and cyanosis recurred and became progressively worse. He was readmitted on Nov. 13, 1952, in marked cardiac decompensation. In spite of vigorous therapy, the patient's condition became worse, and he died on Nov. 15, 1952.

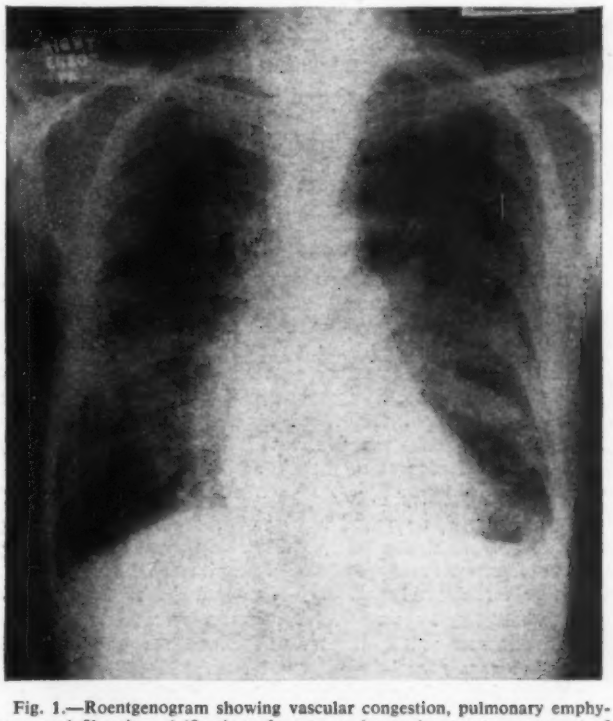


Fig. 1.—Roentgenogram showing vascular congestion, pulmonary emphysema and fibrosis, calcification of aorta, and prominent artery segment of heart in 57-year-old man.

At necropsy the body was emaciated. The pericardial space was obliterated by dense adhesions. Both pleural cavities were also partially obliterated, particularly over the lower and middle lobes. Loculations of clear fluid were present among the adhesions. The heart weighed 645 gm. The left and right ventricles measured 5 and 15 mm. respectively. There was some fibrous thickening of the mitral and tricuspid valves. The coronary vessels were moderately sclerotic but remained patent. The ascending aorta was also sclerotic. The right and left lung weighed 1,450 and 1,150 gm. respectively. There was partial to complete absence of crepitation. The bronchi and bronchial subdivisions were filled with a blood-tinged frothy exudate. The cut surface was blackened, granular, and slightly friable, with patchy areas of fibrosis and consolidation. The liver extended 5 cm. below the xyphoid process and weighed 2,450 gm. The cut surface was moderately congested. The lobular architecture was fairly distinct. The gallbladder and extrahepatic biliary system were normal. The spleen weighed 200 gm. and appeared congested. The gastrointestinal tract and pancreas were not remarkable. The right and left

kidney weighed 240 and 220 gm. respectively. The cut surface was congested and showed some loss of cortical tissue. The rest of the genitourinary tract was normal.

The histological changes of the lungs were most distinctive. There was a diffuse deposition of large quantities of partly hyalinized connective tissue. Many of the alveoli were obliterated. There were very few inflammatory cells, except in the lower lobes where terminal pneumonia had developed. Otherwise, there were a few lymphocytes, plasma cells, and macrophages. The peribronchial and perivascular spaces also showed varying degrees of fibrosis. The bronchial epithelium showed some squamous cell metaplasia. Some of the lung sections from the right upper lobe, as well as the peribronchial lymph nodes, showed anthracosilicosis. There was evidence of passive hyperemia of the other parenchymatous organs, particularly the liver. The cause of death was a combination of congestive failure and terminal pneumonia resulting from the diffuse interstitial fibrosis of the lungs.

COMMENT

The cause of diffuse interstitial fibrosing pneumonitis is obscure. There is no indication from the clinical course or from the laboratory or pathological studies of a bacterial infection or chemical irritation that might be responsible. The histological findings of diffuse interstitial fibrosis with extensive proliferation of connective tissue within the alveolar walls appears to represent a distinct clinicopathological syndrome that, if inflammatory, may represent a type of tissue response to some unknown virus.

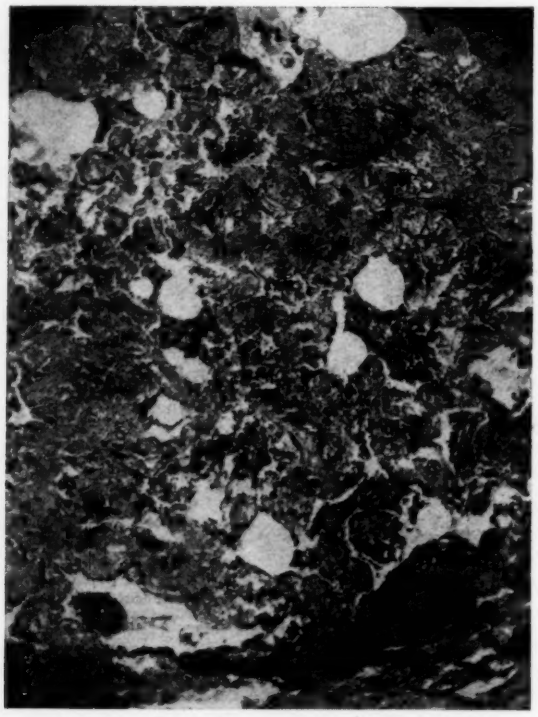


Fig. 2.—Microscopic section taken from lung biopsy of patient with diffuse fibrosing interstitial pneumonitis.

SUMMARY

A case of diffuse fibrosing interstitial pneumonitis is described. In this instance, the diagnosis was made during the patient's life by means of a lung biopsy. Postmortem examination confirmed the findings of the lung biopsy.

1930 Chestnut St. (3) (Dr. Kirshner).

COUNCIL ON PHARMACY AND CHEMISTRY

NEW AND NONOFFICIAL REMEDIES

The following additional articles have been accepted as conforming to the rules of the Council on Pharmacy and Chemistry of the American Medical Association for admission to New and Nonofficial Remedies. A copy of the rules on which the Council bases its action will be sent on application.

R. T. STORMONT, M.D., *Secretary.*

Neomycin Sulfate.—Mycifradin Sulfate (Upjohn).—Neomycin is an antibiotic isolated from the elaboration products of Streptomyces fradiae, when the micro-organism is grown on suitable culture mediums.

Actions and Uses.—Neomycin sulfate is a polybasic compound, thermostable and soluble in water, but insoluble in organic solvents. It differs from other antibacterial agents in that it is extremely stable and very active in alkaline solution. Neomycin is not inactivated by exudates, enzymes, gastrointestinal secretions, and by-products of digestion or bacterial growth. The sulfate salt is stable in the dry state for at least two years when stored at room temperature. Prepared solutions retain their potency for at least one year at room temperature, although there may be a progressive deepening of color of solutions stored at room temperature or 37 C. Refrigeration of neomycin solutions is, therefore, recommended.

Neomycin sulfate exhibits activity against a variety of gram-positive and gram-negative bacteria. In the former group, it appears to be more effective against micrococci (staphylococci) than streptococci. It has a wider antibacterial spectrum than bacitracin, penicillin, or streptomycin, and it is sometimes effective against Pseudomonas and Proteus infections. Micro-organisms resistant to neomycin have been demonstrated in vitro, but emergence of resistant strains has not yet been observed clinically. It may be effective against micro-organisms that have developed resistance to streptomycin; however, the evidence thus far available does not justify the conclusion that neomycin suppresses the overgrowth of resistant bacterial variants. It is not active against fungi.

Neomycin sulfate is useful for topical applications as a solution or ointment in the local treatment or prevention of susceptible infections of the skin and the eye, including pyogenic or secondarily infected stasis dermatoses, impetigo, wounds, burns, ulcers (varicose or trophic), conjunctivitis, blepharitis, and sty (hordeolum). A solution is considered superior to an ointment in treating trophic ulcers and secondarily infected burns. In severe or extensive infections, local therapy should be supplemented with sulfonamides by mouth or penicillin by injection.

Neomycin sulfate is also useful as an intestinal antiseptic by oral administration for suppression of the usual bacterial inhabitants of the colon in surgery of the large bowel and anus. Because of its poor absorption from the gastrointestinal tract, it rarely produces systemic action or toxic effects when administered orally. The small fraction absorbed (about 3% of the amount ingested) is rapidly excreted in the urine; the remainder is excreted unchanged in the feces. Divided total daily oral dosage not exceeding 6 to 10 gm. for one to three days produces blood levels lower than the toxic serum concentration of 0.2 mg. per cubic centimeter that occurs with parenteral injection. Blood levels of this concentration may produce serious kidney damage. *Therefore, neomycin should not be injected parenterally, and when given orally, high dosage and prolonged administration should be avoided to prevent possible systemic effects.* Outgrowth of nonpathogenic yeasts usually follows reduction of the bacteria flora of the colon; Aerobacter aerogenes may grow out about 12 hours after the outgrowth of yeasts. The effectiveness of neomycin is variable in suppressing organisms of the Clostridia group.

Neomycin sulfate is usually well tolerated and is relatively nonirritating. It is reported to have a low index of sensitization. A mild laxative effect occurs with oral administration. Prolonged oral therapy may result in overgrowth of nonsusceptible organisms—particularly Candida. If new infections caused by bacteria or fungi appear during therapy, it may be advisable to discontinue the drug and/or institute appropriate measures to combat them. Oral use as an intestinal disinfectant is contraindicated in the presence of obstruction.

Dosage.—Neomycin sulfate is applied topically or administered orally for its local action in the large intestine.

For external use, it is applied as a solution containing 5 mg. per cubic centimeter or as an ointment containing 5 mg. per gram. The solution is used for wet dressings, packs, irrigations, or instillations. Topical applications of the solution or ointment are made once or twice daily using an amount sufficient to cover the affected region.

For preoperative disinfection of the colon, the patient is placed on a low residue diet and, immediately after the administration of a cathartic (unless otherwise contraindicated), is given an oral dose of 1 gm. every hour for four doses followed thereafter by 1 gm. every four hours for 24 to 72 hours prior to surgery. Administration of the antibiotic should not extend beyond 72 hours. This amount usually produces four to eight bowel movements.

Eli Lilly & Company, Indianapolis.

Ointment Neomycin Sulfate: 14.2, 28.35, and 113.4 gm. tubes. An ointment containing 5 mg. of neomycin sulfate in each gram.

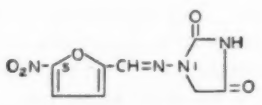
Ophthalmic Ointment Neomycin Sulfate: 3.54 gm. tubes. An ointment containing 5 mg. of neomycin sulfate in each gram.

The Upjohn Company, Kalamazoo, Mich.

Powder Mycifradin Sulfate: Vials containing 0.5 gm. of neomycin sulfate.

Tablets Mycifradin Sulfate: 0.5 gm.

Nitrofurantoin.—Furadantin (Eaton).—$C_8H_6N_4O_5$.—M.W. 238.16.—N-(5-Nitro-2-furfurylidene)-1-aminohydantoin.—The structural formula of nitrofurantoin may be represented as follows:



Actions and Uses.—Nitrofurantoin, a nitrofuran derivative, exhibits a wide spectrum of antibacterial activity against both gram-positive and gram-negative micro-organisms. It is bacteriostatic and may be bactericidal to the majority of strains of Escherichia coli, Micrococcus (Staphylococcus) pyogenes, albus, and aureus, Streptococcus pyogenes, Aerobacter aerogenes, and Paracolobactrum species. The drug is less effective against Proteus vulgaris, Pseudomonas aeruginosa, Alcaligenes faecalis, and Corynebacterium species; many strains of these organisms may be resistant to it; however, bacterial resistance to other anti-infective agents is not usually accompanied by increase in resistance of the organisms to nitrofurantoin. The drug does not inhibit fungi or viruses.

Nitrofurantoin is useful by oral administration for the treatment of bacterial infections of the urinary tract, and its use is indicated in pyelonephritis, pyelitis, and cystitis caused by bacteria sensitive to the drug. It is not intended to replace surgery when mechanical obstruction or stasis is present. After oral administration, approximately 40% is excreted unchanged in the urine. The remainder is apparently catabolized by various body tissues into inactive, brownish compounds that may tint the urine. Only negligible amounts of the drug are recovered from the feces. Urinary excretion is sufficiently rapid to require administration of the drug at four to six hour intervals to maintain antibacterial concentration. The low oral dosage necessary to maintain an effective urinary concentration is not associated with detectable blood levels. The high solubility of nitrofurantoin, even in acid urine, and the low dosage required diminish the likelihood of crystalluria.

Nitrofurantoin has a low toxicity. With oral administration it occasionally produces nausea and emesis; however, these reactions may be obviated by slight reduction in dosage. An occasional case of sensitization has been noted, consisting of a diffuse erythematous maculopapular eruption of the skin. This has been readily controlled by discontinuing administration of the drug. Animal studies, using large doses administered over a prolonged period, have revealed a decrease in the maturation of spermatozoa, but this effect is reversible after discontinuance of the drug. Until more is known concerning its long-term effects, blood cell studies should be made during therapy. Frequent or prolonged treatment is not advised until the drug has received more widespread study. It is otherwise contraindicated in the presence of anuria, oliguria, or severe renal damage.

Dosage.—Nitrofurantoin is administered orally in an average total daily dosage of 5 to 8 mg. per kilogram (2.2 to 3.6 mg. per pound) of body weight. One-fourth of this amount is administered four times daily—with each meal and with food at bedtime—to prevent or minimize nausea. For refractory infections, such as Proteus and Pseudomonas species, the total daily dosage may be increased to a maximum of 10 mg. per kilogram (4.5 mg. per pound) of body weight. If nausea is severe, the dosage may be reduced. Medication should be continued for at least three days after sterility of the urine is achieved.

Tests and Standards.—

Physical Properties: Nitrofurantoin is a yellow, bitter powder with a slight odor, which decomposes at 258-262°. It is very slightly soluble in alcohol, and practically insoluble in ether and water.

Identity Tests: Suspend in a test tube about 5 mg. of nitrofurantoin in 5 ml. of water and add 5 ml. of sodium hydroxide T.S.: the nitrofurantoin dissolves and an orange-red color develops.

Transfer to a 50 ml. Erlenmeyer flask about 50 mg. of nitrofurantoin and add 1 gm. of granular zinc, 10 ml. of alcohol, and 20 ml. of diluted sulfuric acid. Heat the mixture in a steam bath for 45 min.: the nitrofurantoin dissolves slowly to form an almost colorless solution *(presence of nitro group).*

A 0.0005% solution, prepared as described in the spectrophotometric assay for nitrofurantoin, exhibits ultraviolet absorption maxima at about 266 and 368 mμ [specific absorbancy, E(1%,1 cm.), about 753] and a minimum at about 305 mμ. The ratio of the absorbancies at 368 and 266 mμ is 1.26 to 1.46.

Purity Tests: Dry about 1 gm. of nitrofurantoin, accurately weighed, at 105° for 5 hours: the loss in weight does not exceed 1.0%.

Char about 1 gm. of nitrofurantoin, accurately weighed, cool the residue, add 1 ml. of sulfuric acid, heat cautiously until evolution of sulfur trioxide ceases, ignite, cool, and weigh: the residue does not exceed 0.05%.

Assay: (Nitrofurantoin) Prepare a 0.0005% solution of nitrofurantoin as follows. Transfer to a 100 ml. volumetric flask 0.1 gm. of nitrofurantoin, accurately weighed, fill to the mark with dimethylformamide, and mix. Transfer to a second 100 ml. flask 5 ml. of this solution, fill to the mark with water, and mix. Transfer to a third 100 ml. volumetric flask 10 ml. of this last solution, fill to the mark with water, and mix. Spectrophotometrically determine the absorbancy in a 1 cm. quartz cell at 368 mμ, using water as a blank. The concentration of nitrofurantoin in the solution in mg./ml. = absorbancy ÷ 75.3. The amount of nitrofurantoin is not less than 95.0 or more than 105.0%.

Dosage Forms of Nitrofurantoin

TABLETS. *Identity Tests:* Transfer to a test tube about 5 ml. of the clear centrifugate of the first dilution from the spectrophotometric assay for nitrofurantoin in tablets and add 5 ml. of sodium hydroxide T.S.: an orange-red color develops.

The tablets respond to the spectrophotometric identity test for the active ingredient in the monograph for nitrofurantoin.

Assay: (Nitrofurantoin) Weigh 20 tablets and powder them. Transfer to a 100 ml. volumetric flask an amount of powder, accurately weighed, equivalent to about 0.1 gm. of nitrofurantoin, add 50 ml. of dimethylformamide, and shake the flask mechanically for 15 min. Fill to the mark with dimethylformamide and mix. Centrifuge a portion of the solution at 2,000 rpm for 5 minutes. Transfer to a second 100 ml. volumetric flask 5 ml. of the clear centrifugate, fill to the mark with water, and mix. Transfer to a third 100 ml. volumetric flask 10 ml. of this last solution, fill to the mark with water, and mix. Spectrophotometrically determine the absorbancy in a 1 cm. quartz cell at 368 mμ, using water as a blank. The concentration of nitrofurantoin in the solution in mg./ml. = absorbancy ÷ 75.3. The amount of nitrofurantoin is not less than 95.0 or more than 105.0% of the labeled amount.

Eaton Laboratories, Inc., Norwich, N. Y.

Tablets Furadantin: 50 and 100 mg. U. S. patent 2,610,181. U. S. trademark 569,968.

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SMOKER'S RESPIRATORY SYNDROME

Most of the literature on the effect of tobacco on man deals with cardiovascular changes.[1] More recently studies have attempted to show a correlation between smoking and pulmonary carcinoma. Myerson [2] has drawn attention to the effect of excessive smoking on the larynx. Edema is first noted in a localized area of one of the vocal cords. As the condition progresses this becomes bilateral and edematous fibromas are formed. If the condition is of long standing it becomes irreversible and must be treated by surgical removal. Somewhat akin to this but still a separate clinical entity is smoker's respiratory syndrome as described by Waldbott.[3]

This condition is characterized by a triad of symptoms consisting of chronic pharyngitis, wheezing and dyspnea, and a tendency to respiratory infection with or without fever. The pharynx is fiery red and may be covered with a mucopurulent discharge. Small lymphoid nodules form on the wall of the pharynx. Other symptoms include cough, a sense of constriction in the chest, chest pain, and hoarseness. The wheezing may be explained by the fact that the mucus formed by the chronic inflammation partially obstructs the air passages. The presence of mucus and the bronchoconstrictor action of nicotine account for the sense of constriction in the chest, the bronchospasm, and the dyspnea.

This condition is very common and is often mistaken for bronchial asthma or bronchiectasis. Unlike asthma this syndrome has no allergic basis; the wheezing is confined to the trachea and the upper portion of the chest; emphysema, if present, is slight; there is no eosinophilia; and the vital capacity is only slightly decreased. Some patients may have both asthma and the respiratory syndrome.

Early cases are reversible and require only the elimination of smoking to effect a cure within about seven days. The symptoms return if smoking is resumed. More advanced cases require extensive antibiotic treatment in addition to the avoidance of tobacco smoke, and a few are so far advanced as to be irreversible. The recognition of this condition is important in order to prevent subjecting the patient to troublesome and unnecessary allergic testing or visualization of the bronchial tree and to remove the cause before advanced changes take place.

1. Waldbott, G. L.: Smoker's Respiratory Syndrome, A Clinical Entity, J. A. M. A. **151**: 1398-1400 (April 18) 1953.
2. Myerson, M. C.: Smoker's Larynx: A Clinical Pathological Entity, Ann. Otol. Rhin. & Laryng. **59**: 541-546 (June) 1950.
3. Waldbott, G. L.: Further Observations on Smoker's Respiratory Syndrome, Ann. Int. Med. **39**: 1026-1031 (Nov.) 1953.

FOURTEENTH ANNUAL CONGRESS ON INDUSTRIAL HEALTH

There is increasing interest in stress as a factor in the development of disease. Industrial physicians and others engaged in the physical examination of executives have been especially concerned. The so-called quasi-occupational diseases, such as hypertension, peptic ulcer, colitis, obesity, alcoholism, and others, have been under observation for long periods of time without clear-cut understanding and weighing of the effects of nonoccupational as opposed to occupational tensions and situations. There is good evidence that these stress disorders are not more prevalent in executive or supervisory groups than they are in the working population generally.

This matter will receive serious attention from clinical, geriatric, and pyschosomatic points of view during the 14th annual Congress on Industrial Health at the Brown Hotel in Louisville, Ky., February 24 and 25. Closely associated will be discussions on keeping well persons well, to include estimations of the present health status of industrial workers, plans for making physical examinations more effective in industry and in the community, and a practical demonstration of the preventive medical program now in operation in a large American corporation.

Emphysema is a clinical condition frequently encountered by internists, chest physicians, radiologists, and industrial practitioners. This subject will be thoroughly explored in its clinical aspects and with regard to pathological physiology, histopathology, radiography, and therapy. Certain developments in the occupational health field make it imperative that the etiological factors bearing on emphysema be well understood.

A special feature of the sessions on February 24 will be the presentation of the Physician's Award for 1953 for an outstanding contribution to the employment welfare of the handicapped. This award, signed by President Eisenhower, is made by the President's Committee on Employment of the Physically Handicapped.

Most of the first day sessions will be devoted to keeping employees at work, a subject of first-rank importance in periods of rising absenteeism. A panel will present an optimal industrial health and safety program with emphasis on small plant participation. Since first aid and emergency follow-up are always prime elements in an effective prevention program, instructions and demonstrations in this field will be provided by the national and local Red Cross organizations.

The final half day will be conducted jointly with the Council on National Emergency Medical Service, with able assistance from the Federal Civil Defense Administration. The principal components in major disaster will be dramatically described. The program will conclude with a practical demonstration of preparation for trouble in Vicksburg, Miss., and how industry and medicine proved to be an indispensable team when a major catastrophe struck that city.

The program of the Congress appears in this issue of THE JOURNAL. The Brown Hotel is holding space for those requesting reservations for the meeting. Reservations should be made promptly.

IRON ABSORPTION IN HEMOCHROMATOSIS

Hemochromatosis is a metabolic disturbance characterized clinically by diabetes mellitus, cirrhosis of the liver, and skin pigmentation. In addition, there is extensive deposition of iron throughout the body. Apparently in this condition there is increased absorption of iron from the gastrointestinal tract; since the body has no mechanism for disposing of other than insignificant amounts of iron, increased intake leads to accumulation in the tissues. Evidence in support of this viewpoint has come from balance studies with radioactive iron, which is particularly useful in studies of this type in view of the difficulty of recovering iron quantitatively from feces and tissues in the standard colorimetric procedures and because of the many possibilities of introducing extraneous iron as a contaminant.

A recent study is that of Peterson and Ettinger.[1] These investigators administered 5 to 15 μ of Fe^{59} as ferric chloride orally to two patients with hemochromatosis and to four normal subjects. Ferric iron was chosen because it is absorbed less readily than ferrous iron and because dietary iron probably exists in the ferric form. It was administered to fasting subjects to avoid factors in the diet known to interfere with the absorption of iron. The radioactivity of the iron in the blood serum was measured one, two, and three hours after administration and that of the red blood cells at intervals of 10, 15, and 20 days. Stools were collected for one to two weeks for analysis of unabsorbed iron. The gastrointestinal absorption could be calculated from the amount not recovered in the feces since only minute amounts of iron are excreted into the gastrointestinal tract. The absorption of iron in the normal subjects ranged from 1.5% to 4.4% while the uptake by the red blood cells at the end of 15 days ranged from 1% to 4% of the ingested dose. In the two patients with hemochromatosis, the absorption was approximately 20% and 45% respectively and the uptake in the red blood cells some 16% of the ingested dose. Increased iron absorption in the patients with hemochromatosis was also indicated by the fact that radioactive iron was present in appreciable amounts in their serum but was indetectable in the serum of all the controls.

It can be seen that in one patient the amount of iron absorbed was three times as great as that found in the red blood cells. The authors felt that this discrepancy arose from the fact that when excessive amounts of iron are stored in the liver only a small portion of that recently absorbed will go to the production of red blood cells. Support for this viewpoint is offered by the fact that the patient in whom the amount of iron absorbed corresponded to the amount taken up by the red blood cells had undergone repeated phlebotomies, which had served to reduce his iron stores. Thus, the uptake of radioiron by red blood cells cannot be used as a reliable index of iron absorption in the presence of excessive iron storage; in such cases balance studies provide the most accurate information.

The report by Peterson and Ettinger is in accord with previous studies by two other groups of investigators. Dubach, Callender, and Moore [2] reported that a patient with hemochromatosis absorbed about 20% of an oral dose of radioiron as ferrous chloride, while only 2% appeared in his blood. While the amount absorbed was considerably greater than that in most of the normal control subjects, the amount in the blood was considerably less, thus emphasizing the erroneous results that may be obtained if the amount of radioiron in the circulatory hemoglobin is taken as a measure of iron absorption. Alper, Savage, and Bothwell,[3] giving radioiron as ferrous sulfate, found that a patient with hemochromatosis absorbed about 60% and a normal control subject about 15% of the administered dose. In the patient, the radioiron uptake in the blood reached an initial peak about three hours after administration, amounting to about 1.5% of the dose. It then fell off sharply and rose again slowly to a peak of about 4% about three months later. Apparently the radioiron was removed from the plasma, stored, and gradually built into hemoglobin.

While increased iron absorption in hemochromatosis appears to be well established, it remains to be determined what the factors are that modify the absorption and whether the increased iron stores in the tissues play a primary role in this disease. Evidence that excessive iron storage does have a deleterious effect is indeed suggested by the clinical improvement that has been reported to follow depletion of iron stores by repeated massive phlebotomies.[4]

REFERRED CARDIAC PAIN

In 1910 Hirschfelder [1] described an arrangement of the dermatomes involved in referred pain, derived from the work of Head [2] and Mackenzie.[3] It was suggested that pain from the right ventricle is referred to the right hypochondrium and that pain from the left auricle is referred to the left hypochondrium. Great progress has been made since the turn of the century. Clinical experience now affirms that in most instances angina is referred to the left substernum and arm, regardless of whether its origin is in the left or right ventricle or in the ventricular septum. The complex winding and unwinding of the cardiac tube in the course of its ontogeny has introduced complexities that are still not completely understood.

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2. Dubach, R.; Callender, S. T., and Moore, C. V.: Studies in Iron Transportation and Metabolism: VI. Absorption of Radioactive Iron in Patients with Fever and with Anemias of Varied Etiology, Blood **3**: 526, 1948.
3. Alper, T.; Savage, D. V., and Bothwell, T. H.: Radioiron Studies in a Case of Hemochromatosis, J. Lab. & Clin. Med. **37**: 665, 1951.
4. Peterson and Ettinger,[1] Davis, W. D., Jr., and Arrowsmith, W. R.: The Treatment of Hemochromatosis by Massive Venesection, Ann. Int. Med. **39**: 723, 1953.
1. Hirschfelder, A. D.: Diseases of the Heart and Aorta, Philadelphia, J. B. Lippincott Company, 1910.
2. Head, H.: On Disturbances of Sensation, with Special Reference to the Pain of Visceral Disease, Brain **16**: 1-133, 1893.
3. Mackenzie, J.: Some Points Bearing on the Association of Sensory Disorders and Visceral Disease, Brain **16**: 321-354, 1893.

The common territory of pain reference forms a band broader at the midsternum, where it extends from the third or fourth to the sixth or seventh intercostal spaces; it continues anteriorly across the upper axilla almost bisecting the ventral aspect of the arm and remains limited to the inner area, narrowing as it approaches the wrist to taper off on the ventral surface of the fifth finger.[4] Occasionally the distribution is right-sided, but this is not related to an origin in the right ventricle. Uncommon zones of radiation include the head, scalp, face, neck, clavicular regions, and shoulder. The aorta is sometimes represented, as in dissecting aneurysm, by a radiation to the scapula, but commonly the pain is severe, difficult to localize, and easily confused with anginal pain. Pain from the pulmonary artery is not clinically understood.

The auricles are certainly hindmost in the development of the cardiac tube and theoretically should receive their innervation from more caudal portions of the thoracic segments. Actually the complex structure of the superficial and right and left branches of the deep cardiac plexuses and their numerous connections and intricate pathways have not yet provided the simple, logical, and satisfying pattern that was envisioned by our scientific forebears. On the basis of the recent work of Prof. A. Barry of Ann Arbor, a more adequate knowledge of the derivation of the great arteries has become available.[5] The pulmono-aortic trunk derives from the former ventral aorta and is represented by cervical segments. The main pulmonary arterial branches derive from the sixth aortic arch, while a small segment of the transverse aorta comes from the sixth arch. The segment of the aorta giving rise to the subclavian artery comes from the primitive third to seventh segments, and the descending aorta is derived from successive segments beginning with the eighth. The aortic and pulmonary innervation is thus rather complex and differs significantly from the interpretations of earlier workers.

HEPATOMEGALY AND DIABETES

The liver frequently is increased in size in diabetics. This has been recognized for many years. In fact, special attention was called to this phenomenon in children with severe uncontrolled diabetes mellitus as long ago as 1938.[1] More recently, Goodman [2] found that 21 of 45 diabetics with hepatomegaly had an insulin defficiency as evidenced by poorly regulated diabetes or acidosis; 13 were obese and only mildly diabetic, and 11 had a lipotropic deficiency as determined by their dietary history. Histological examinations have shown that the increase in the size of the liver is due to fatty deposits, not to glycogen, and control of the diabetes causes disappearance of the hepatomegaly, thus suggesting that the underlying cause of the hepatomegaly is poor diabetic control and not a lack of choline or other derivatives of raw pancreatic tissue.

Goodman now finds that, because the liver may be palpable without being enlarged and vice versa, palpation is an unreliable criterion of true hepatomegaly. Furthermore the palpation of an enlarged liver in an obese patient is difficult. He therefore advocates estimating liver enlargement by percussing the extent of the liver dulness in the midclavicular line and places the upper limit of normal at 7.5 cm. in men and at 7 cm. in women.[3]

In order to determine whether there was a correlation between the hepatomegaly in diabetics and the state of control of the diabetes he observed a series of patients that included (1) those with controlled diabetes, (2) those who passed 50 or more grams of glucose in their urine in 24 hours, and (3) those with ketoacidosis. Hepatomegaly was present in 9% of the first group, 60% of the second, and all of the third. The livers in the third group were greatly enlarged and tender. The hepatomegaly disappeared after about 10 days with a regimen that controlled the diabetes, or if the patients were obese the liver regained its normal size with a return to normal body weight.

The author concluded that such symptoms as polyuria and polydipsia and the presence of hepatomegaly or neuropathy are better indications of the state of diabetic control than the amount of glucose in a 24-hour specimen of urine. He believes that the best possible liver function should be maintained in diabetics and that diabetic control furthers this end.

ELECTRICAL CHARGES ON PERSONS AND AUTOMOBILES

During periods when the humidity is very low, such as on cold, dry days, there is a tendency for persons and objects to develop a static electrical charge on the surfaces. This tendency is enhanced by friction, as when one slides over a car seat or walks across a rug. A moving automobile readily develops an electrical charge due to the friction of the air flowing over its surface. More or less discomfort arises when a person touches another person or some object and the static charge discharges.

Relief from this situation can be brought by humidification of the air in the room, but this does not, of course, solve the problem when this phenomenon occurs out of doors. One way of minimizing the development of a static charge on the surface of an automobile is to attach a piece of "conductive rubber" to the car so that the conductive rubber drags on the ground. This will allow for discharge of the electrostatic charge. Persons who find the phenomenon quite disturbing can avoid this difficulty by touching the object from which it is suspected a shock will result with something metallic such as a coin or a key gripped tightly in the hand. Under these conditions electrical discharge passes between the metal coin or key and the object the person is about to touch, so that the person does not experience the discomfort caused by the electrical discharge.

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1. Marble, A.; White, P.; Bogan, I. K., and Smith, R. M.: Enlargement of the Liver in Diabetic Children: I. Its Incidence, Etiology and Nature, Arch. Int. Med. **62**: 740-750 (Nov.) 1938. White, P.; Marble, A.; Bogan, I. K., and Smith, R. M.: Enlargement of the Liver in Diabetic Children: II. Effect of Raw Pancreas, Betaine Hydrochloride and Protamine Insulin, ibid. **62**: 751-764 (Nov.) 1938.

2. Goodman, J. I.: Enlarged Liver in Diabetes Mellitus: Its Determination by Percussion, Am. J. Digest. Dis. **18**: 181-185 (June) 1951.

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ORGANIZATION SECTION

FOURTEENTH ANNUAL CONGRESS ON INDUSTRIAL HEALTH

Brown Hotel, Louisville, Ky.

Feb. 24-25, 1954

Sponsored by

The Council on Industrial Health of the American Medical Association

Sessions are open to physicians and others interested in industrial health

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PROGRAM

WEDNESDAY MORNING, FEB. 24, 9:30

OPENING GENERAL SESSION

J. DUFFY HANCOCK, M.D., Louisville, *Presiding*

Industry and Medicine—Partners in Health
ROBERT A. KEHOE, M.D., Cincinnati

Panel Discussion—Stress and Success—A Medico-Social Problem of Modern American Life

Moderator:
WILLIAM P. SHEPARD, M.D., New York

Participants:
EDWARD L. BORTZ, M.D., Philadelphia
LEO H. BARTEMEIER, M.D., Detroit

WEDNESDAY AFTERNOON, FEB. 24, 2:00

SECTION I. KEEPING WELL PEOPLE WELL

LEMUEL C. MCGEE, M.D., *Presiding*
Wilmington, Del.

Are Workers Getting Healthier?
FRANK DICKINSON, PH.D., Chicago

Making Physical Examinations More Effective in Industry and the Community
Speaker to be announced

The Preventive Medical Program of the du Pont Company
ALLAN J. FLEMING, M.D., Wilmington, Del.
GEORGE W. HOGSHEAD, M.D., Nitro, W. Va.
ALFRED W. PENNINGTON, M.D., Wilmington, Del.
FREDERICK W. DERSHIMER, M.D., Wilmington, Del.

SECTION II. EMPHYSEMA—OCCUPATIONAL ASPECTS OF A COMMON CLINICAL FINDING

O. A. SANDER, M.D., Milwaukee, *Presiding*

Clinical Aspects
GEORGE W. WRIGHT, M.D., Cleveland

Pathological Physiology
WARD S. FOWLER, M.D., Rochester, Minn.

Histopathology
MARVIN KUSCHNER, M.D., New York

Radiography
LEONARD J. BRISTOL, M.D., Saranac, N. Y.

Therapy
FRANK PRINCI, M.D., Cincinnati

WEDNESDAY EVENING, FEB. 24, 6:30

ANNUAL DINNER

With the Jefferson County Medical Society

DAVID M. COX, M.D., Louisville, *Presiding*

National Award for 1953—To Physician Making an Outstanding Contribution to the Employment Welfare of the Handicapped

Rehabilitation in Louisville
J. MURRAY KINSMAN, M.D., Louisville

Who's Disabled?—A Dramatic Presentation of What Is Left —Not What Is Lost
DONALD A. COVALT, M.D., New York, Demonstrator

THURSDAY MORNING, FEB. 25, 9:00

Panel Discussion—Keeping People at Work—A Small Plant Operator Consults the Experts

Moderator:
H. F. REINHARD, New York

Participants:
WARREN A. COOK, PH.D., Ann Arbor, Mich.
GAY MERRIHUE, R.N., New York
JOHN GIBSON, Washington, D. C.
CARL OLSON, M.D., Chicago
GENE COLCORD, Owensboro, Ky.

The Plant Prepares for Emergencies
Demonstrations of First Aid and Follow-Up Procedure arranged with assistance from the Louisville Chapter, American Red Cross

THURSDAY, FEB. 25, 12:00

LUNCHEON

With Louisville Rotary Club

L. EUGENE JOHNSON, Louisville, *Presiding*

Address
VAL PETERSON, Washington, D. C.

THURSDAY AFTERNOON, FEB. 25, 2:00

JOINT SESSION WITH THE COUNCIL ON NATIONAL EMERGENCY MEDICAL SERVICE

JAMES SARGENT, M.D., Milwaukee, *Presiding*

IF MAJOR DISASTER STRIKES

Atomic Bombing
CHARLES DUNHAM, M.D., Washington, D. C.

Bacterial Warfare
JOHN J. PHAIR, M.D., Cincinnati

Chemical Warfare
COL. JAMES DEFANDORF, Washington, D. C.

Psychological Warfare
OZRO T. WOODS, M.D., Dallas, Texas

HOW WE FACED UP TO DISASTER

Preparation and Organization
LAWRENCE LONG, M.D., Jackson, Miss.

Industry and Medicine—The Indispensable Disaster Team
GEORGE MARTIN, M.D., Vicksburg, Miss.

Local Application
NEIL DALTON, Louisville

SUMMARY OF THE CONFERENCE

ANOTHER RECORD YEAR FOR AMERICAN MEDICAL EDUCATION FOUNDATION

With the close of business on Dec. 31, 1953, the American Medical Education Foundation chalked up some additional "firsts" in its effort to raise funds for our country's medical schools. The foundation, working through its 53 state and metropolitan committees, recorded a gross return of $1,090,-771.13 from 17,809 individual physicians, organizations, and laymen. This is the first time since the inception of the foundation that its annual income has passed the million dollar mark. Foundation officials believe that this will have a marked effect on the foundation's future returns, since it evidences growing acceptance among the medical profession. Also worthy of note and indicative of future success is the continuing growth of contributors, which increased 149% over 1952 (17,809 compared to 7,259 in 1952). There has been a marked increase in dollar contributions received from individuals as compared to previous years. Approximately $475,000 of the total received in 1953 came from this source, and it amounted to a 63% increase over the same income segment for 1952.

Illinois took an early lead in the state standings and held the lead throughout the entire year, largely due to the introduction of the "Illinois Plan," whereby the House of Delegates to the Illinois State Medical Society voted a blanket increase of $20 per member in the state dues, which were turned over to the foundation. Utah has recently followed the example set by Illinois and introduced the same method. Leaders in Illinois who support this method of raising funds believe that it will solve the knotty problem of organizing and maintaining active committees for the purposes of annual solicitation of the society's membership. It is a plan worthy of study by states having a heavy physician population where the maintenance of active committees poses a problem. The passage of the dues increase actually stimulated the income from individual physicians in that state over the previous year. Illinois physicians gave $12,121 and the Chicago Medical Society made a treasury gift of $25,000 to bring the total income from Illinois in 1952 to $37,121. The Illinois plan produced $167,630 from 8,386 dues-paying members. In addition to the dues increase group, 86 contributions were received from Illinois, totaling $22,831.99 When compared to 1952, Illinois moved from 35th place in relative state standings (88 contributors gave $37,121 in 1952) to first place in the nation, with a total of 8,472 contributors and total contributions of $190,461.99 during 1953. We believe that the Illinois plan acted as a stimulus in Illinois and that such a plan would undoubtedly serve similarly in other states. It is a plan worthy of consideration.

The American Medical Association voted a fourth grant of $500,000 to the foundation to be paid during the calendar year of 1954. As in the case of the past three grants in the same amounts, it will serve as seed money for the 1954 campaign for funds among the profession. The American Medical Association has given a total of 2 million dollars in four successive grants to this project since December, 1950.

State chairmen of the American Medical Education Foundation will meet on Jan. 24, 1954, at the Sheraton Hotel in Chicago to discuss campaign plans for the current year. The one day conference will afford state chairmen an opportunity to discuss and exchange problems and suggestions beneficial to their respective local campaigns.

The American Medical Education Foundation has enjoyed steady progress throughout its three year history and has contributed approximately 50% of the $4,764,152.79 that has been distributed to the nation's medical schools through the grants made by the National Fund for Medical Education. Recent decisions handed down by the supreme court of the state of New Jersey have removed obstacles standing in the way of corporations that wished to make donations to education. With the medical profession's attainment of a 2 million dollar annual goal, it is believed that American business and industry will produce willingly contributions necessary to meet and relieve the financial problems of our medical schools. The American Medical Education Foundation deserves the support of every physician in the United States. It has one purpose—giving financial assistance to our institutions of medical education—the same institutions that have consistently graduated the world's best-trained physicians.

Every member of the medical profession can look with pride at the compact group of 79 approved medical schools in this country; nowhere in the world can another nation boast their equal. This system of medical education is a by-product of a nation of free people, working together to produce the world's highest health standards for their own benefit. It is a well-known fact that a medical student does not pay the full cost of his medical education. A substantial part of that cost must come from the general funds of the parent university. Therefore, it behooves all members of the medical profession to remember their obligation to the school where they received their medical training and to contribute financially each year in proportion to their means. If every member of the medical profession will resolve to make a contribution this year, the foundation will more than meet its quota during 1954.

JOINT COMMISSION ON ACCREDITATION OF HOSPITALS

The Director of the Joint Commission on Accreditation of Hospitals, Dr. Edwin L. Crosby, has forwarded the following report of the annual meeting of the Board of Commissioners of the Joint Commission, which is sponsored by the American College of Physicians, American College of Surgeons, American Hospital Association, American Medical Association, and the Canadian Medical Association.

The annual meeting of the Board of Commissioners of the Joint Commission was held on December 5 in Chicago. The officers for 1954 are Newell W. Philpott, M.D., Chairman; LeRoy H. Sloan, M.D., Vice-Chairman; Stuart K. Hummel, Treasurer, and Edwin L. Crosby, M.D., Secretary. The Board expressed its gratitude and sincere appreciation to Gunnar Gundersen, M.D., as Charter Chairman. His leadership and wise counsel in this initial period have given the Joint Commission a firm base and a good beginning toward attaining its purpose of supporting better patient care.

Consultations

The Board received a report on the "Consultation" Standard from a review committee composed of Doctors Philpott, Chairman, Alexander M. Burgess and Stanley R. Truman. The Committee recommended and the Board approved the following as Standard Required II B 3:

"Except in emergency, consultation with a member of the Consulting or of the Active medical staff shall be required in all major cases in which the patient is not a good risk, or in which the diagnosis is obscure, and in all first caesarean sections, sterilizations, curettages or other operations which may interrupt a known, suspected, or possible pregnancy. The consultant shall make and sign a record of his findings and recommendations in every such case. In all cases where a rule of the hospital requires consultation, the consultant may, and in the case of free patients shall, give his services without charge. This Standard is applicable to all members of the medical staff."

The Board asked the Committee on Consultations to continue its study and report back after July 1, 1954. The Committee would appreciate comments or suggestions from any interested individual or group as to the Standard on Consultations as stated in this Bulletin which, since the Board action of December 5, should be followed by hospitals and their medical staffs.

Interpretation of Dental Staff Standard

The Board reviewed the recommendations of a liaison committee between representatives of the Joint Commission and the American Dental Association headed by Dr. Leslie M. Fitzgerald, President. The Committee considered Standard Contingent II D. "Dental Staff."

"The dental staff shall conform in general to standards established for the medical staff (see Required II). In addition:

1. Members of the dental staff shall be graduates of approved schools of dentistry.

2. Patients admitted for dental services shall be admitted on a surgical service and shall be the responsibility of the chief of that service.

3. Adequate medical survey, by a member of the service to which admitted, shall be done on each patient before dental surgery. Indicated consultations shall be held in complicated cases."

The Board approved the following interpretation of this Standard with regard to the dental staffs in well departmentalized hospitals:

"Depending upon the local situation and the qualifications of the individuals involved, Contingent II D, Section 2, might be interpreted to mean that 'a surgical service' may be the oral surgical service to which the members of the dental staff admit and discharge dental patients, provided that this service is the responsibility of the Chief of the Department of Surgery and that Section 3 of the Standard is complied with actively.

"In these instances, the bylaws of the staff would indicate that the staff of the hospital would be a 'medical and dental staff' and that within the framework of the hospital's staff bylaws, dentists would be appointed to and hold appointments in the division of oral surgery just as physicians and surgeons are appointed to other services of the hospital."

The Board emphasized that "the medical survey" referred to in Contingent II D 3 must be done by a Doctor of Medicine on the medical staff of the hospital.

Smaller Hospitals

The Board recognized that smaller hospitals have special problems in regard to accreditation, largely in the matter of interpretation of the Standards. The Chairman agreed to appoint a special committee to review this situation and devise methods to assist the smaller hospitals.

General Practice

The Board passed a resolution commending the American Academy of General Practice for its initiative in developing the Manual on General Practice Departments in Hospitals as a guide in the organization of such departments.

On December 6, representatives of the Joint Commission met with members of the Commission on Hospitals of the American Academy of General Practice. The group discussed the underlying principles of medical staff meetings, as published in the August Bulletin of the Joint Commission.

Such staff meetings are held for the purpose of a thorough review of the medical care of patients, in or recently discharged from the hospital, and frequent meetings of clinical departmental staffs are regarded as essential in well-organized departmentalized hospitals.

After the discussions, the following was stated as proper:

In a well departmentalized hospital electing to hold at least monthly clinical departmental meetings and quarterly general medical staff meetings in accord with Contingent II, A 4 b, a doctor who holds an "active staff" appointment in a Department of General Practice should attend at least nine (75% of 12) departmental meetings per year held by the clinical services in which he has privileges in addition to 75% attendance at the quarterly meetings of the entire active medical staff. One of the two following patterns for attendance at clinical departmental meetings should be followed by "an active staff member" of a Department of General Practice:

1) If he has privileges as an "active staff member" in a clinical department, he should attend 75% of the monthly meetings of that department just as all other active staff members of that clinical department. It is understood that no physician will have "active staff" appointment in more than one clinical department.

OR

2) If he has privileges other than as an "active staff member" in clinical departments, he should attend at least nine departmental meetings per year among the clinical services in which he has such privileges. The clinical departmental meetings attended shall be of his own choice, with the exception that he might be requested to attend a particular departmental clinical meeting.

1,175 Hospitals Surveyed

The Director reported that during the first eleven months of 1953, the Board had acted on 1,175 hospital surveys. The Commission began actual survey operations January 1, 1953. A listing of Fully and Provisionally Accredited Hospitals as of December 31, 1953 will be published early in 1954.

The Board was informed that the field staff had observed the following factors affecting patient care as the major deficiencies noted during their surveys:

1. Fire hazards.

2. Need for improvement in the active supervision of the clinical work done in the hospital by a well organized medical staff which is self-governing, subject to the ultimate authority of the governing body of the hospital.

3. Need for improvement in the thorough review, analysis, and evaluation of the clinical work done in the hospital on at least a monthly basis throughout the year.

4. Insufficiently recorded essential clinical entries on the medical records to establish the diagnosis and support the treatment.

5. Excessively high and unexplained rates for morbidity, Caesarean Sections, "not justified" removal of "normal tissue" and infant, maternal, anesthetic and total mortality in a hospital.

As we approach the end of our first year of operations, the great interest and voluntary participation in hospital accreditation of the hospitals and their medical staffs in the United States and Canada is a source of satisfaction and gratification to the Board of Commissioners and the staff of the Joint Commission on Accreditation of Hospitals. Many of you have questions concerning details of our program. Please write to the Commission's office at 660 North Rush Street, Chicago.

REGIONAL LEGISLATIVE CONFERENCES

The A. M. A. Committee on Legislation is completing plans for six regional legislative conferences to be held in various sections of the country. These meetings will have a two-fold purpose: (1) to explain and perfect the system now being followed for alerting key legislative personnel in the states to situations requiring immediate contact with members of Congress on national legislative affairs and (2) to discuss in detail background information regarding important medical issues pending or coming up in the new Congress.

The schedule of the legislative meetings is as follows: Saturday, Jan. 23, in San Francisco; Sunday, Jan. 24, Denver; Saturday, Jan. 30, Dallas, Texas; Sunday, Jan. 31, Atlanta, Ga.; Saturday, Feb. 6, Chicago; and Saturday, Feb. 13, New York. Those invited to the meetings are the chairmen and members of legislative committees of state medical societies and other key legislative men in the states. States to be represented at the San Francisco meeting are Oregon, California, Idaho, Nevada, and Washington; at Denver—Arizona, Colorado, Utah, Wyoming, Montana, Nebraska, North Dakota, South Dakota, Kansas, and Iowa; at Dallas—Arkansas, Louisiana, New Mexico, Oklahoma, and Texas; at Atlanta—Alabama, Florida, Georgia, North Carolina, South Carolina, Missouri, Mississippi, Tennessee, and Kentucky; at Chicago—Indiana, Illinois, Michigan, Minnesota, and Wisconsin; and at New York —Maine, Vermont, Massachusetts, New Hampshire, Rhode Island, Ohio, Pennsylvania, West Virginia, New York, Delaware, Connecticut, Maryland, New Jersey, and Virginia.

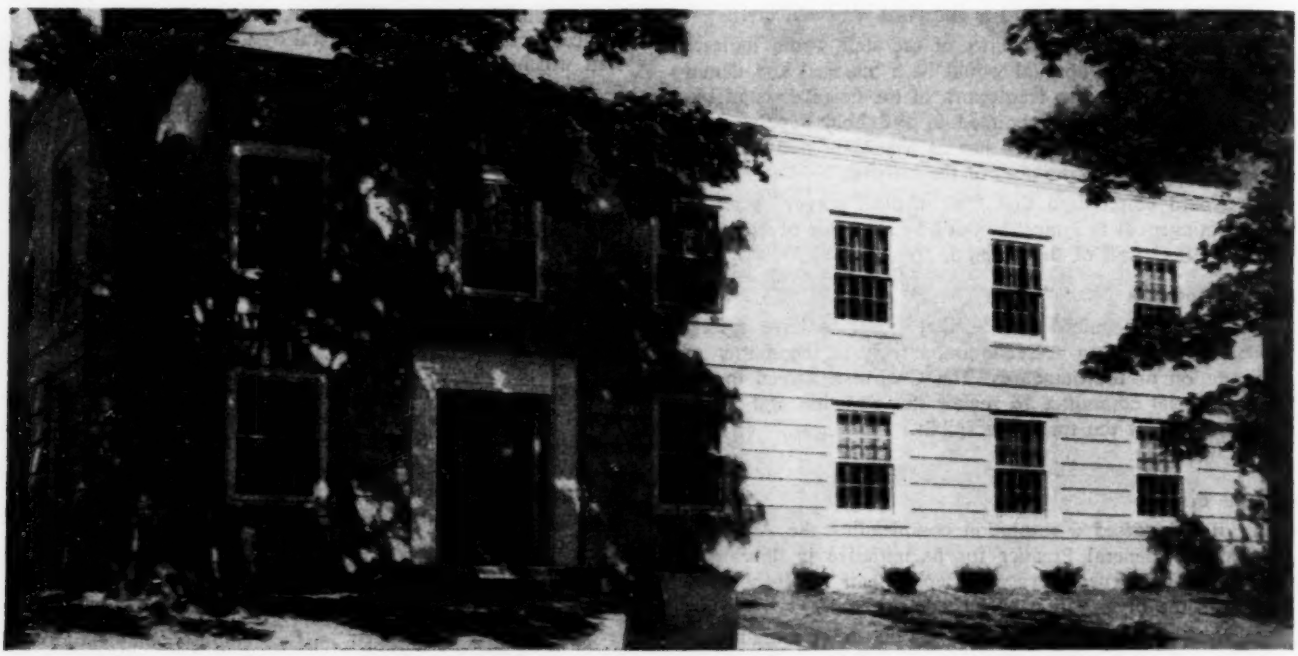
CONNECTICUT STATE MEDICAL SOCIETY

Under what is believed to be the first private charter granted by the Connecticut legislature, the Connecticut State Medical Society was incorporated in May, 1792. Long before the formation of a state society, however, groups of physicians had banded together to form medical organizations in various counties—New London, New Haven, and Litchfield—the records dating back to a meeting for that purpose in 1739 in New Haven. In February, 1780, a meeting was held in Sharon to form the "First Medical Society in the Thirteen United States of America since their Independence," but it was not until Jan. 5, 1784, that the New Haven County Medical Association was founded at a meeting called for the purpose of uniting "in petition with other county societies to the honorable legislative body for an act of incorporation the more effectually to regulate the practice of physic." A number of years and petitions later, the society finally found a way to establish the sincerity and integrity of its aims and eventually to succeed. In January, 1788, it voted that a collection of the most interesting cases that had been communicated to the society be made and that they be published in a pamphlet at the expense of the society. "Cases and Observations," said to be the first medical transactions published in America, acted as such a stimulus that a library was established, a librarian appointed, and the New Haven County society encouraged anew to seek a charter as an educational institution.

The petition for the incorporation of the Medical Society of New Haven was again filed, in January, 1791, and again defeated; but the following year, through a petition centering attention on the improvement in the quality of medical education and service, the goal was reached. A charter was bestowed that gave the society the privilege to grant degrees in medicine, a privilege that it exercised until 1813, after the society had joined with the corporation of the Yale College to found the "Medical Institution of Yale College." Through the first half century of its history this institution was administered jointly by the society and the college, the society being largely responsible for choice of the faculty, for examining all candidates, and for determining those who should receive medical degrees. Each constituent county society was allowed to name a student to attend the school without charge.

Early in its course and pursuant to a communication on the care of lunatics, presented by one of its members, the society, under the leadership of its president, Dr. Mason Fitch Cogswell, initiated a crusade for the public care of the insane and in 1821 launched a project for the establishment of a hospital for the insane, voting some of its own funds for this purpose. As a result, the Hartford Retreat, still in operation as the Institute of Living, was chartered by the legislature. During these years Dr. Cogswell was instrumental also in incorporation of the Connecticut Asylum for the Education and Instruction of Deaf and Dumb Persons, the first school for the education of the deaf in America. The Hartford Retreat having been established, the society turned its attention to plans for a general hospital for the state, "to be so located as to best serve the interests of the Medical Institution of Yale College," and "Old North" was established in 1831.

Recent public service activities of the society include direction of a movement to create the Commission on the Chronically Ill, Aged, and Infirm (1945) and a State Commission on Mental Health (1953). In June, 1942, the society observed the sesquicentennial celebration of its founding. In commemoration of this occasion a book, "The Heritage of Connecticut Medicine," was published under the editorship of Dr. Herbert Thoms. It contains a host of great names in Connecticut medicine, including those of Drs. Jared Eliot, Elisha North, Nathan Smith, Horace Wells, Lyman Hall, William Beaumont, Jared P. Kirtland, William H. Carmalt, and William Henry Welch.



Headquarters of Connecticut State Medical Society.

The society occupies its own building, built by contributions from its members in 1949 and containing such historical paintings as "The New Haven Coffee House," scene of the founding of the New Haven County Medical Association, from which the state society stemmed; "The Medical Institution of Yale College"; "The First Building of the Hartford Retreat"; "The General Hospital of Connecticut"; and "The Birthplace of William Beaumont." The Connecticut Hospital Association, the Connecticut Medical Examining Board, and the State Advisory Committee to the Selective Service also have offices in the building.

The society's 2,875 members constitute more than 90% of the physicians in Connecticut. Its officers are Dr. George H. Gildersleeve, Norwich, president; Dr. Harold M. Marvin, New Haven, president-elect; Dr. Thomas J. Danaher, Torrington, chairman of the council; Dr. Frank H. Couch, Cromwell, treasurer; and Dr. Creighton Barker, New Haven, executive secretary.

MEDICAL NEWS

ALABAMA

Cancer Seminar.—Under the joint sponsorship of the American Cancer Society, Alabama division, the Medical Association of the State of Alabama, Medical College of Alabama, Jefferson County Dental Association, and the Jefferson County Medical Society, a cancer seminar will be held at the Tutwiler Hotel, Birmingham, Jan. 27-28. On Wednesday the following presentations will be made:

Role of the Dentist in Early Recognition of Oral Cancer, Frank F. Kanthak, Atlanta, Ga.
Use of the Proctoscope in Detection of Cancer, Michael R. Deddish, New York.
Detection of Gastrointestinal Neoplasms, Ross Golden, New York.
Detection of Early Carcinoma in the Female Genital Tract, John A. Wall, Houston, Texas.
Evaluation of Present-Day Diagnosis and Treatment of Prostatic Cancer, William W. Scott, Baltimore.
Fibrocystic Disease of the Breast as an Obstacle to the Early Detection of Breast Cancer, Frank E. Adair, New York.

A cocktail hour, 6 p. m., will precede the dinner, at which Dr. William Boyd, University of British Columbia, Vancouver, Canada, will present "Some Thoughts on Carcinogenesis." The Thursday program includes:

Neoplastic Diseases in Childhood, John R. Schenken, Omaha.
Early Detection of Cancer of the Lung, Richard H. Overholt, Brookline, Mass.
Early Stages, Recognition and Treatment of Cancer of the Skin, Richard L. Sutton Jr., Kansas City, Mo.

CALIFORNIA

Stanford Appoints Medical Deans.—Dr. Windsor Cooper Cutting, faculty member of Stanford University School of Medicine, San Francisco, for 15 years and acting dean since July, 1953, has been appointed dean of the school. Dr. Jay Ward Smith has been named associate dean for professional services and Dr. George Bernard Robson associate dean for academic affairs. Dr. Cutting, a Stanford alumnus, succeeds Dr. Loren R. Chandler who, after serving for 20 years, resigned as dean in October, 1952, but continues as professor of surgery. Dr. Cutting has done research on virus diseases and antibiotics and is the author of two books and many papers in professional journals. He was named executive head of the department of pharmacology and therapeutics in 1950. Dr. Smith, who will be medical supervisor of Stanford University Hospitals, including the clinical services and laboratories, will also have charge of postgraduate medical training. He received his medical degree from Stanford in 1944 and served two years in the Army Medical Corps. While in practice in Millbrae, he was clinical instructor at Stanford Medical School, and for the past two years has been in Indianapolis doing research for a pharmaceutical firm and teaching medicine at the Indiana University School of Medicine. Dr. Robson received his medical degree from Stanford in 1934 and has been on the clinical teaching staff of the school for 16 years. As associate dean for academic affairs, he will be responsible for undergraduate medical instruction. He has been in private medical practice in San Francisco and served in the Army Medical Corps from 1943 to 1946.

COLORADO

Dr. Stead to Present Sewall Lectures.—Dr. Eugene A. Stead Jr., professor of medicine, Duke University School of Medicine, Durham, N. C., will present the annual Henry J. Sewall lectureship in the Denison auditorium, University of Colorado School of Medicine, Denver, Jan. 28. The lectureship, established in 1937, memorializes Dr. Henry J. Sewall, who came to Colorado in 1898 as professor of physiology at the Denver and Gross Medical College, served as professor of medicine at the University of Colorado from 1911 to 1918, and was professor emeritus from 1920 until his death in 1936. In 1887 Dr. Sewall discovered the principle of active immunity following inoculation with rattlesnake venom, pointing the way to the development of diphtheria and other antitoxins.

Members of the medical profession have been invited to attend the conferences and laboratory discussions which Dr. Stead will conduct at the school of medicine during the entire week of Jan. 25.

Physicians are invited to send to this department items of news of general interest, for example, those relating to society activities, new hospitals, education and public health. Programs should be received at least three weeks before the date of meeting.

CONNECTICUT

Dr. Gallivan Honored.—Dr. John N. Gallivan, East Hartford, chief medical supervisor of the United Aircraft Corporation, whose leadership has created productive employment for more than 12,000 handicapped workers, has been cited for outstanding service by the President's Committee for Employment of the Physically Handicapped. The citation was presented to Dr. Gallivan at the semiannual meeting of the Connecticut State Medical Society's House of Delegates, Dec. 10, in New Haven. Dr. Gallivan's record of service for the handicapped began in June, 1937, when he became assistant chief medical supervisor of United Aircraft Corporation. A year later he was promoted to his present position and assumed responsibility for industrial health programs at the corporation's four main operating plants. Dr. Gallivan is a member of the Connecticut Committee for Employment of the Physically Handicapped and health director for civil defense in East Hartford. He is president of the East Hartford Medical Society, member of the board of directors of the Hartford County Medical Association, and the association's representative on the Council of the Connecticut State Medical Society. He is a member and past chairman of the state medical society's committee on industrial health and chairman of the society's committee on insurance.

GEORGIA

Ophthalmologists and Otolaryngologists Meet in Savannah.—The annual meeting of the Georgia Society of Ophthalmology and Otolaryngology will be held at General Oglethorpe Hotel, Wilmington Island, Savannah, March 5-6. The Friday program will open at 9 a. m. with the movie "Otoscopic Cinematography of the Tympanic Membrane and Middle Ear" by Dr. Paul H. Holinger, Chicago, and the Saturday program at the same time with the movie "Reconstruction of Canaliculi After Trauma and Disease" by Dr. Alston Callahan, Birmingham, Ala. Guest lecturers and their first presentations include:

Lawrence R. Boies, Minneapolis, Serous Effusion. Chronic Suppurations.
Francis E. LeJeune, New Orleans, Management of Malignancies of the Larynx.
James H. Maxwell, Ann Arbor, Mich., Diseases of the Salivary Glands.
Paul A. Chandler, Boston, Complications of Cataract Extraction.
Algernon B. Reese, New York, Causes and Treatment of Defective Vision in Children.
Henry P. Wagener, Rochester, Minn., Types of Retinal Arterial Occlusion: Their Diagnosis and Treatment.

On Friday, 6:30 p. m., cocktails in the Sunrise Lounge will precede a smorgasbord in the main dining room, which will be followed by General Oglethorpe's famed turtle races in the Crystal Ballroom. Registration fee, $20.

ILLINOIS

Cancer Grant.—At the annual meeting of the Illinois division of the American Cancer Society, a check for $41,250 was accepted, on behalf of the University of Chicago by Dr. Charles B. Huggins, professor of surgery. The money, which is the first payment on a $165,000 institutional grant, will be used to support principally a vast clinical research program, but some help will also be given to the continuing basic research program. Dr. Huggins spoke on scientific research in cancer.

Chicago

Naval Medical Unit Programs.—On Jan. 26, 8 p. m., Dr. Matthew Taubenhaus will discuss "Hormonal Regulation of Granulation Tissue Formation" at the meeting of Volunteer Medical Company 9-20 at the U. S. Naval Reserve Armory, Randolph St. and the lake. The same group will be addressed Feb. 2 by Dr. Frederick Steigmann, who will speak on liver profile, and on Feb. 9 by Dr. Samuel L. Goldberg, whose topic will be hyperparathyroidism. On Feb. 23 Dr. Townsend B. Friedman will present "Immune-Chemistry of Allergic Disease."

Biochemistry of Pathological Processes.—The University of Chicago announces a course in recent developments in biochemistry at University College, 19 S. LaSalle St., Wednesdays, 7:30 p. m. Lectures will be given on the anemias (Jan. 27), neoplastic growth: local biochemistry (Feb. 3) and systematic biochemical effects (Feb. 10), radiation (Feb. 17), and dental disease (Feb. 24). On March 3, Dr. Morris A. Lipton, assistant professor and resident in medicine (psychiatry), will deliver a lecture on mental disease. Endocrine disorders will be discussed by Dr. Delbert M. Bergenstal, assistant professor of medicine, March 10, and a discussion of electrolyte balance in health and disease will be offered by Lillian Eichelberger, Ph.D., associate professor in biochemistry and surgery on March 17.

IOWA

Second Heart Institute.—The second Institute on Cardiovascular Diseases sponsored by the Speakers Bureau of the Iowa State Medical Society, the Iowa Heart Association, and the State Department of Health, in cooperation with the State University of Iowa College of Medicine, Iowa City, is to be held at the Veterans Administration Hospital, Des Moines, Jan. 29. The afternoon program, 3-5 p. m., will be devoted to a consideration of vascular hypertension, with Dr. Arthur C. Corcoran, of the Cleveland Foundation, as principal speaker. The evening program, 7:30-9:30 p. m., will concern peripheral vascular disease, and Dr. Carl A. Moyer, St. Louis, will give the main address. A social hour will precede dinner at 6:30 p. m. The third Institute on Heart Diseases is to be held in Sioux City in May.

LOUISIANA

Appoint Professor of Dermatology.—Dr. Charles B. Kennedy, who has been affiliated with the Louisiana State University School of Medicine, New Orleans, since 1934, has been named professor of dermatology to replace the late Dr. James K. Howles as head of the department. During World War II Dr. Kennedy was with the 64th General Hospital, the school of medicine unit, in Africa and Italy. He was awarded the Bronze Star medal by the Mediterranean Theater Commander for writing a history of skin diseases and syphilis in this area and for organizing clinics. He formerly served as an assistant dermatologist at the Columbia University College of Physicians and Surgeons, New York, for three years and has been a visiting physician at Charity Hospital of Louisiana.

MASSACHUSETTS

Meeting on Heart Disease.—Dr. Paul D. White, Boston, will serve as moderator for the following program on the management of heart disease, which the Norfolk district of the Massachusetts Medical Society will present at 8:15 p. m., Jan. 26, in the Boston Medical Library (Ware Hall), 8 The Fenway:

Benedict F. Massell, Boston, Rheumatic Heart Disease and Subacute Bacterial Endocarditis.
Louis Wolff, Boston, Coronary Heart Disease.
Gordon S. Myers, Boston, Consideration of Surgery for Heart Disease.

Course on Pulmonary Function.—A postgraduate course on the measurement of pulmonary function in health and disease, sponsored by the American Trudeau Society in cooperation with the Massachusetts Tuberculosis and Health League, Harvard Medical School, Boston University School of Medicine, and Tufts College Medical School, Boston, will be held in March at the Boston City Hospital. Registration is limited. The fee is $50, payable to the Massachusetts Tuberculosis and Health League. A limited number of scholarships covering partial or total expenses will be available through the courtesy of the National Tuberculosis Association. Subjects include mechanical and physiologic aspects of respiration, methods of analysis of pulmonary function, cardiopulmonary relationships, alterations of pulmonary function as a result of disease, alterations of pulmonary function as a result of therapy, and clinical application of tests of pulmonary function. Physicians accepted for this course may obtain information regarding hotel accommodations from Mr. Nelson R. Kraemer, Executive Director, Massachusetts Tuberculosis and Health League, 131 Clarendon St., Room 802, Boston 16.

MICHIGAN

Carl E. Buck Dies.—Carl E. Buck, Dr. P. H., professor of public health, University of Michigan School of Public Health, and formerly field director of the American Public Health Association, died in Ann Arbor, Nov. 21, 1953, aged 62. Dr. Buck had been on the staff of the School of Public Health, after 17 years of service as field director. He had participated in scores of public health studies, principally at the state level, and had directed the Colorado state survey that resulted in a reorganization of the state department of health. Between 1924 and 1930 Dr. Buck held several positions in the Detroit Department of Health. He was a member of the 1912 U. S. Olympic team and during World War I was a pilot in the U. S. Air Corps. After the war he was appointed a member of the U. S. Typhus Fever Commission in the Balkan countries. On Nov. 11, 1953, the American Public Health Association bestowed on Dr. Buck, former president, Michigan Public Health Association, the Sedgwick memorial award.

MINNESOTA

Annual Johnston Lecture.—Andrew T. Rasmussen, Ph.D., professor emeritus of neuroanatomy, will present the annual J. B. Johnston lecture Jan. 27 at the University of Minnesota Center for Continuation Study. His subject will be "Recent Advances in the Morphology and Significance of the Cerebral Cortex."

Society News.—Newly elected officers of the Southwestern Minnesota Medical Society include Dr. Roy F. Pierson, Slayton, president, Dr. Roger P. Hallin, Worthington, president-elect, Dr. Harold A. Christensen, Jackson, vice-president, and Dr. Olaf M. Heiberg, Worthington, secretary (reelected).

Chamber of Commerce Honors Dr. Sherman.—Dr. Charles L. Sherman, who has practiced in Luverne for 50 years, was guest of honor at a reception given by the Luverne Chamber of Commerce, Sept. 24, 1953, the evening being designated as "Dr. Sherman Night." The program included a talk by Dr. E. Mendelssohn Jones, St. Paul. The Southwestern Minnesota Medical Society adopted a resolution expressing high praise and deep appreciation for the service Dr. Sherman has rendered to humanity and the profession of medicine in his community and state.

MISSOURI

Case of Epidemic Hemorrhagic Fever.—According to the U. S. Public Health Service, Dr. James R. Amos, director, Missouri Department of Public Health and Welfare, has reported epidemic hemorrhagic fever in a 24-year-old man. The patient left a highly endemic area of the disease in Korea, Nov. 14, 1953, arrived by air in the United States four days later, and was almost immediately discharged from military service. There is no evidence of person-to-person transmission of epidemic hemorrhagic fever, and the arthropod suspected of transmitting infection is not known to be present in the United States.

NEW JERSEY

Symposium on Diseases of the Liver.—A symposium on diseases of the liver, stressing the treatment of cirrhosis and its complications, ascites and esophageal varices, will be given at the Jersey City Medical Center, Jan. 27, 2-5 p. m. Speakers will include Drs. Arthur H. Blakemore, John L. Madden, and J. James Smith, New York, Carroll M. Leevy, Jersey City, and Jacob K. Berman, Indianapolis.

Postgraduate Lectures at Cohanzick Country Club.—The postgraduate education committee of the Cumberland County Medical Society will present the following lectures by faculty members of the University of Pennsylvania School of Medicine, Philadelphia, at the Cohanzick Country Club, 2 miles south of Bridgeton on the Fortescue-Port Norris Road:

Jan. 26, 9:30 p. m.., Early Management of Obscure Fever in Children, Milton Rapoport.
Feb. 16, 4 p. m., Management of Cardiac Emergencies, Charles C. Wolferth.
March 16, 4 p. m., Diagnosis and Management of Gastric Lesions, Isidor S. Ravdin.

Each lecture will be followed by a discussion period. The cost of the series is $10. Information may be obtained from Dr. W. Sherman Garrison Jr., 108 W. Commerce St., Bridgeton.

NEW YORK

Public Cancer Forum.—"The Curability of Cancer" was the subject of the public cancer forum recently conducted by the Yonkers Academy of Medicine at the Commerce High School auditorium in Yonkers. Support was given by the Westchester Cancer Committee and other professional and social agencies, and the Yonkers Board of Education passed an endorsing resolution and approved the public forum as a part of its adult education program. Religious groups of all denominations also helped publicize this forum, as did the Yonkers Chamber of Commerce and the CIO.

University News.—Dr. Curtland Chester Brown Jr., Glenmont, has been appointed assistant dean of the Albany Medical College. In addition to administrative duties, Dr. Brown holds an instructorship in the department of medicine and is a member of the staff of the Albany Hospital.——The University of Buffalo has appointed Dr. Samuel Feinstein chief psychiatrist of the Chronic Disease Research Institute and medical director of therapy of the Information and Rehabilitation Center for Alcoholism. Dr. Feinstein has been affiliated with the New York State Department of Mental Hygiene, the St. Lawrence State Hospital and A. Barton Hepburn Hospital, Ogdensburg, and New York State Teachers College, Potsdam.

New York City

Another Unit in Medical Center.—The new Medical Science Building of the New York University-Bellevue Medical Center, now under construction between 13th and 34th streets and First Avenue and the Franklin D. Roosevelt Drive, is scheduled for completion by early summer. This building will accommodate classrooms and teaching laboratories for N. Y. U. College of Medicine and its Post-Graduate Medical School; complete research laboratories for all departments of the schools and for the various institutes of the medical center; and other facilities. The Medical Science Building is the second new building of a $28,500,000 medical center serving New York and the surrounding community. The Institute of Physical Medicine and Rehabilitation is completed and in operation. Still to be constructed are Alumni Hall, the Hall of Residence, and the 20-story, 600-bed University Hospital.

NORTH CAROLINA

Personal.—Dr. Edward C. Frank, associate professor of psychiatry at the University of North Carolina School of Medicine, Chapel Hill, has entered private practice in Louisville. Dr. Frank was formerly adjunct psychiatrist at the Psychosomatic Institute at Michael Reese Hospital, psychiatrist for the David B. Rotman Research Laboratory of the Psychiatric Institute of the Municipal Court of Chicago, and consultant to Portal House outpatient clinic, Chicago, and the alcoholic unit of the mental health centers of the state of Illinois. His new address is Francis Building, Louisville.

Free Transportation for Eye Bank.—The Eye Bank for Restoring Sight, Inc., of North Carolina, chartered by the state in 1951 to secure and supply eyes to all North Carolina hospitals and physicians, is an affiliate of the Eye Bank for Sight Restoration, Inc., of New York. Three members of the Medical Society of the State of North Carolina form an advisory committee, and each airport town in the state has a regional medical director who aids in disseminating knowledge about the eye bank. Eyes are carried free of charge by Piedmont Airlines, Eastern Airlines, Capital Airlines, and by private taxi and limousine services in the state.

SOUTH CAROLINA

Symposium on Cardiovascular Disease.—The South Carolina Heart Association in conjunction with the Greenville General Hospital and Greenville County Medical Society will present a symposium on cardiovascular disease, Feb. 1. Participants will include Dr. Robert P. Grant, National Heart Institute, Bethesda; Dr. Leslie B. Hohman, professor of psychiatry, Duke University School of Medicine, Durham, N. C.; Dr. Harold Feil, clinical professor of medicine, Western Reserve University School of Medicine, Cleveland; and Dr. Elliott V. Newman, professor of experimental medicine, and Dr. H. William Scott Jr., professor of surgery and surgeon-in-chief, Vanderbilt University School of Medicine, Nashville, Tenn. Drs. Feil and Scott will be the after-dinner speakers Monday evening, when the Greenville County Medical Society holds its monthly meeting in the ballroom of the Poinsett Hotel.

VIRGINIA

Fifty Year Club.—Certificates carrying life membership were recently awarded by the Medical Society of Virginia to the following physicians, who graduated 50 years ago: Joseph LeCenne DeCormis, Accomac; Courtney Edmond, Clifton Forge; William Wallace Gill, Richmond; William H. Howard, Hampton; Louis Knight Leake, Goochland; William Cline Moomaw, Bedford; Lawson Otwood Powell, Seaford; Marvin Pierce Rucker, Richmond; John Bonney Shipp, Norfolk; Benjamin Clinton Shuler, Shenandoah; Homer Amos Spitler, Middleburg; Harry Benjamin Stone, Roanoke; William Joshua Sturgis, Nassawadox; Charles Walter Thomas, Floyd; Edward Victor Valz, Philadelphia; Cary Elphus Via, Norfolk; A. McGarvey Wallace, Gate City; and Elbert Patton Whited, Honaker.

WISCONSIN

Society News.—Officers elected at the recent meeting of the Wisconsin Society of Anesthesiologists include Dr. Robert K. Dodge, Janesville, president, Dr. Paul E. Campbell, Waukesha, president-elect, and Dr. William R. Kreul, Racine, secretary-treasurer.

Course on Surgical Considerations in General Practice.—This course will be conducted under the direction of Dr. Erwin R. Schmidt, professor of surgery, University of Wisconsin Medical School, Feb. 9-11 at State of Wisconsin General Hospital. The course will stress preoperative and postoperative care, general surgical procedures, the relationship of surgery to anesthesiology, antibiotic therapy, and the presence of complicating conditions. The course will be limited to 40 registrants. Advance registration is required, and $5 registration fee must be forwarded to Dr. Robert C. Parkin, University of Wisconsin Medical School, 418 N. Randall Ave., Madison 6. The balance of the fee ($15) may be paid at the time of registration, Feb. 9. Make all checks payable to the University of Wisconsin.

GENERAL

Psoriasis Research Association.—The recently organized Psoriasis Research Association has announced as its first project a survey of 2,000 cases of psoriasis, to be conducted with the voluntary help of physicians and statisticians from the University of California Hospital, San Francisco. The association, a nonprofit organization, has a threefold program: (1) to encourage and finance research, (2) to keep the membership informed of the progress of the association's program and that of the medical profession, by sending periodic bulletins to each member, and (3) to stimulate public education concerning psoriasis. Membership is open to all persons with psoriasis and to everyone interested in this work (annual fee $5). The organization does not conduct research but will award research grants on recommendation of its medical advisory board, consisting of

Drs. Eugene M. Farber, San Francisco, Maximilian E. Obermayer, Los Angeles, Thomas H. Sternberg, Los Angeles, and Frances A. Torrey, San Francisco. Headquarters of the foundation are at 44 Page St., San Francisco 2.

Fellowships in Endocrinology.—The Endocrine Society has announced its 1954 program of awards and fellowships. Each member may make one nomination for each fellowship or award. A nomination should be accompanied by a statement of the importance of the nominee's contribution to endocrinology and by a bibliography of his most important publications, with reprints if possible. Nominations should be made on special application forms which may be obtained from the secretary, Dr. Henry H. Turner, 1200 N. Walker St., Oklahoma City, and returned to him not later than March 1. The Ayerst, McKenna and Harrison fellowship is offered in the amount of $5,000 and the Ciba award in the amount of $1,800, with the provision that, if within 24 months of the date of the award, the recipient chooses to use it toward further study in a laboratory other than that in which he is at present working, it will be increased to $2,500. This award is offered to an investigator, not more than 35 years of age, in the field of clinical or preclinical endocrinology. The Schering Fellowship in Endocrinology will not be awarded in 1954 in order that it may accumulate toward a $5,000 fellowship to be awarded in 1955. Fellowships may be awarded to a person who possesses a Ph.D. or an M.D. degree or to a candidate for either of these degrees. Each candidate for fellowship must (1) present evidence of scientific ability as attested by studies completed or in progress and/or the recommendation of responsible persons, (2) submit a program of proposed study, (3) indicate one or more institutions where the proposed program will be followed, and (4) submit statements of approval from the investigators with whom he proposes to conduct his research. He must serve full time if awarded a fellowship. A small amount of time (10-15%) may be alloted for course work or for participation in teaching, the latter purely on a voluntary basis.

Lasker Public Health Awards.—At a recent meeting of the American Public Health Association the following winners of the 1953 Lasker awards were announced: Michael Heidelberger, Ph.D., professor of immunochemistry, Columbia University College of Physicians and Surgeons, New York, "for decisive contributions to mankind in developing a new subscience, the precise measuring tool of immunochemistry"; Dr. Hans A. Krebs, professor of biochemistry, University of Sheffield, England, "discoverer of the urea and citric acid cycles which are basic to our understanding of how the body converts food into energy"; Earle B. Phelps, B.S., (who died shortly after his election for the award), research engineer at the University of Florida, Gainesville, "in recognition of a lifetime of pioneering and leadership in public health and sanitary science"; Dr. Felix J. Underwood, Jackson Miss., state health officer of Mississippi for 30 years, "for demonstrating how a long-sustained, sound, and expanding pattern of public health services benefits a people"; and George Wald, Ph.D., professor of biology, Harvard University, Cambridge, "in recognition of his outstanding achievements in explaining the chemistry of vision in man."

Group awards were given to the Division of Research Grants of the National Institutes of Health, U. S. Public Health Service, "for the outstanding administration of a research grants program enabling thousands of capable scientists in hundreds of institutions to contribute knowledge substantially advancing the nation's health" and to the University Laboratory of Physical Chemistry Related to Medicine and Public Health, Harvard University, "for basic protein studies leading to fundamental achievements in the field of blood separation, and more particularly, in gamma globulin preparation."

Individual winners received cash prizes of $1,000, leather-bound citations, and gold statuettes of the Winged Victoria of Samothrace. Group winners received silver statuettes. The Lasker awards are made annually through the American Public Health Association by the Albert and Mary Lasker Foundation.

LATIN AMERICA

Seminars in Medicine of Mexico.—Following the annual meeting of the American College of Physicians in Chicago, April 5-9, a series of lectures and presentations is being arranged at the National Institute of Cardiology of Mexico and at the Hospital de Enfermedades de la Nutricion. The scientific programs will be presented during the mornings, April 12-14. The schedule calls for departure from Chicago at 10:40 a. m., April 10, by DC-6 Flagship of the American Airlines, with arrival at Mexico City at 5:35 p. m. The price, $386.15, includes: round trip air transportation from Chicago; double room with twin beds and bath at all hotels, two in room; all meals outside Mexico City, and all breakfasts, lunches, and other meals as specified in the city; sightseeing as detailed in private automobiles; English speaking guides and admissions; all tips when on tours and all tips to porters and bellmen for handling baggage; U. S. Federal tax; Mexico air ticket tax; local taxes; Mexico tourist card; and seminar fee. Single room is $17.75 additional. For information, write to Leon V. Arnold, 36 Washington Square West, New York 11.

FOREIGN

German Course in Tropical Medicine and Hygiene.—A course in tropical medicine, tropical hygiene, pathology, and medical parasitology will be held Feb. 8 to April 10 at the Bernard Nocht Institute for Ship and Tropical Diseases in Hamburg. Physicians, veterinarians, and zoologists are eligible to attend. Information on the course can be obtained from the institute, Bernhard-Nocht Strasse 74, Hamburg 4, Germany.

CORRECTION

Endameba Histolytica.—In the Clinical Note "Leukopenia Following Fumagillin Treatment for Amebiasis," in THE JOURNAL, Dec. 19, 1953, page 1446, in the eighth line in the Report of Case, the term Escherichia histolytica is erroneous and should be Endameba histolytica.

MEETINGS

AMERICAN MEDICAL ASSOCIATION: Dr. George F. Lull, 535 North Dearborn St., Chicago 10, Secretary.
1954 Annual Meeting, San Francisco, June 21-25.
1954 Clinical Meeting, Miami, Florida, Nov. 30-Dec. 3.
1955 Annual Meeting, Atlantic City, N. J., June 6-10.
1955 Clinical Meeting, Boston, Nov. 29-Dec. 2.
1956 Annual Meeting, Chicago, June 11-15.

ANNUAL CONGRESS ON INDUSTRIAL HEALTH, Brown Hotel, Louisville, Ky., Feb. 24-25. Dr. Carl M. Peterson, 535 N. Dearborn St., Chicago 10, Secretary.

ANNUAL CONGRESS ON MEDICAL EDUCATION AND LICENSURE, Palmer House, Chicago, Feb. 7-9. Dr. Edward L. Turner, 535 N. Dearborn St., Chicago 10, Secretary.

NATIONAL CONFERENCE ON RURAL HEALTH, Baker Hotel, Dallas, Texas, Mar. 4-6. Mrs. Arline Hibbard, 535 N. Dearborn St., Chicago 10, Secretary.

AMERICAN ACADEMY OF ALLERGY, Shamrock Hotel, Houston, Texas, Feb. 1-3. Dr. Alan G. Cazort, 1425 West 7th St., Little Rock, Ark., Secretary.

AMERICAN ACADEMY OF FORENSIC SCIENCES, Drake Hotel, Chicago, Feb. 25-27. Prof. Ralph F. Turner, Michigan State College Dept. of Police Administration, East Lansing, Mich., Secretary.

AMERICAN ACADEMY OF OCCUPATIONAL MEDICINE, John Marshall Hotel, Richmond, Va., Feb. 11-12. Dr. Leonard J. Goldwater, 600 W. 168th St., New York 32, Secretary.

AMERICAN ACADEMY OF ORTHOPAEDIC SURGEONS, Palmer House, Chicago, Jan. 23-28. Dr. John R. Norcross, 122 S. Michigan Blvd., Chicago 3, Secretary.

AMERICAN COLLEGE OF RADIOLOGY, Drake Hotel, Chicago, Feb. 5. Mr. William C. Stronach, 20 N. Wacker Drive, Chicago 6, Executive Secretary.

AMERICAN FEDERATION FOR CLINICAL RESEARCH:
SOUTHERN SECTION, Jung Hotel, New Orleans, Jan. 29. Dr. William Parson, University of Virginia School of Medicine, Charlottesville, Va., Chairman.
WESTERN SECTION, Auditorium, University of Oregon Medical School, Portland, Ore., Jan. 28. Dr. I. S. Edelman, University of California Medical Center, San Francisco Hospital, San Francisco 15, Secretary.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY: WESTERN SECTION, University of Oregon Medical School, Portland, Ore., Feb. 6. Dr. Guy L. Boyden, 1735 N Wheeler Ave., Portland 12, Ore., Chairman.

AMERICAN ORTHOPSYCHIATRIC ASSOCIATION, Hotel Commodore, New York, March 11-13. Dr. Marion F. Langer, Room 210, 303 Lexington Ave., New York 16, Executive Secretary.

AMERICAN PROTESTANT HOSPITAL ASSOCIATION, Palmer House, Chicago, Feb. 10-12. Mr. Albert G. Hahn, Protestant Deaconess Hospital, Evansville, Ind., Executive Secretary.

AMERICAN RADIUM SOCIETY, The Homestead, Hot Springs, Va., March 14-16. Dr. Robert E. Fricke, 102 Second Ave. S.W., Rochester, Minn., Secretary.

CENTRAL SURGICAL ASSOCIATION, Statler Hotel, Detroit, Feb. 18-20. Dr. Robert M. Zollinger, University Hospital, Columbus 10, Ohio, Secretary.

CHICAGO MEDICAL SOCIETY ANNUAL CLINICAL CONFERENCE, Palmer House, Chicago, March 2-5. Dr. Maurice M. Hoeltgen, 86 East Randolph St., Chicago, Secretary.

CONFERENCE OF TEACHERS OF CLINICAL RADIOLOGY, Walton Room, Drake Hotel, Chicago, Feb. 6. Dr. Aubrey O. Hampton, 20 North Wacker Drive, Chicago, Chairman.

DALLAS SOUTHERN CLINICAL SOCIETY, Dallas, March 15-18. Dr. T. Haynes Harvill, 433 Medical Arts Bldg., Dallas 1, Texas, Secretary.

MICHIGAN CLINICAL INSTITUTE, Sheraton-Cadillac Hotel, Detroit, March 10-12. Dr. L. Fernald Foster, 606 Townsend St., Lansing 15, Mich., Secretary.

MID-SOUTH POST GRADUATE MEDICAL ASSEMBLY, Hotel Peabody, Memphis, Tenn., Feb. 9-12. Dr. Thurman Crawford, 869 Madison Ave., Memphis, Tenn., Secretary.

NATIONAL CONFERENCE ON CARE OF THE LONG-TERM PATIENT, Edgewater Beach Hotel, Chicago, March 18-20. Dr. Dean W. Roberts, 615 N. Wolfe St., Baltimore 5, Director.

NATIONAL CONFERENCE ON TRICHINOSIS, A. M. A. Headquarters, Chicago, March 1. Dr. S. E. Gould, Wayne County General Hospital, Eloise, Mich., Chairman.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS, Jefferson Hotel, St. Louis, March 10-12. Dr. F. M. Foote, 1790 Broadway, New York 19, Executive Director.

NORTHWEST SOCIETY FOR CLINICAL RESEARCH, Portland, Ore., Jan. 28. Dr. Arthur L. Rogers, 1216 S.W. Yamhill St., Portland, Ore., Secretary.

REGIONAL MEETINGS, AMERICAN COLLEGE OF PHYSICIANS:

TOPEKA, KANS., March 19. Dr. William C. Menninger, 3617 West 6th Ave., Topeka, Kansas, Governor.

RICHMOND, VA., Feb. 25. Dr. Charles M. Caravati, 807 West Franklin St., Richmond 20, Va., Governor.

SOUTHERN CALIFORNIA, Riverside, Feb. 13-14. Mr. E. R. Loveland, 4200 Pine St., Philadelphia 4, Executive Secretary.

SECTIONAL MEETINGS, AMERICAN COLLEGE OF SURGEONS:

CHARLOTTE, N. C., Hotel Charlotte, Feb. 1-3. Dr. Thomas D. Sparrow, 403 North Tryon St., Charlotte 2, N. C., Chairman.

FRENCH LICK SPRINGS, Indiana, French Lick Springs Hotel, March 15-17. Dr. Carl H. McCaskey, 20 N. Meridian St., Indianapolis 4, Chairman.

RENO, NEV., Riverside Hotel, Feb. 25-26. Dr. Kenneth F. Maclean, 120 N. Virginia St., Reno, Nev., Chairman.

OMAHA, NEB., Hotel Fontenelle, March 1-4. Dr. Earl A. Connolly, 107 South 17th St., Omaha, Chairman.

SOCIETY OF UNIVERSITY SURGEONS, Sheraton Hotel, Rochester, N. Y., Feb. 11-13. Dr. C. Rollins Hanlon, 1325 South Grand Blvd., St. Louis, Secretary.

SOUTHEASTERN SURGICAL CONGRESS, Tutwiler Hotel, Birmingham, Ala., March 8-11. Dr. Benjamin T. Beasley, 45 Edgewood Ave. S.E., Atlanta, Ga., Secretary.

WESTERN SOCIETY FOR CLINICAL RESEARCH, Portland, Ore., Jan. 29-30. Dr. Herbert N. Hultgren, Stanford Hospital, San Francisco 15, Secretary.

WESTERN SOCIETY OF ELECTRO-ENCEPHALOGRAPHY, Del Monte Lodge, Pebble Beach, Calif., March 7-8. Dr. Sylvester N. Berens, 902 Boren Ave., Seattle, Secretary.

FOREIGN

ASSOCIATION OF SURGEONS OF GREAT BRITAIN AND IRELAND, Leeds, England, May 13-15, 1954. Dr. Henry W. S. Wright, 45 Lincoln's Inn Fields, London W.C.2, England, Honorary Secretary.

BRITISH MEDICAL ASSOCIATION, Glasgow, Scotland, July 1-9, 1954. Dr. A. Macrae, B.M.A. House, Tavistock Square, London, W.C.1, England, Secretary.

CANADIAN MEDICAL ASSOCIATION, Vancouver, B. C., Canada, June 14-18, 1954. Dr. T. C. Routley, 244 St. George St., Toronto 5, Ont., Canada, General Secretary.

CONFERENCE OF INTERNATIONAL UNION AGAINST TUBERCULOSIS, Madrid, Spain, Sept. 26-Oct. 2, 1954. Secretariat, Escuela de Tisiologia, Ciudad Universitaria, Madrid, Spain.

CONGRESS OF INTERNATIONAL ANESTHESIA RESEARCH SOCIETY, Los Angeles, Calif., U. S. A., Oct. 4-7, 1954. For information write: Dr. T. H. Seldon, 102-110 Second Avenue S. W., Rochester, Minn., U. S. A.

CONGRESS OF INTERNATIONAL ASSOCIATION FOR THE PREVENTION OF BLINDNESS, New York, N. Y., U. S. A., Sept. 12-17, 1954. Professor Franceschetti, 2 Avenue Mirmot, Geneva, Switzerland, Secretary-General.

EUROPEAN SOCIETY OF CARDIOVASCULAR SURGERY, Edinburgh, Scotland, July 9-10, 1954. For information address: Mr. A. J. Slessor, Department of Surgery, University New Building, Edinburgh 8, Scotland.

HEALTH CONGRESS OF THE ROYAL SANITARY INSTITUTE, Scarborough, England, April 27-30, 1954. Mr. P. Arthur Wells, Royal Sanitary Institute, 90 Buckingham Palace Road, London, S.W.1, England, Secretary.

INTERNATIONAL CANCER CONGRESS, Sao Paulo, Brazil, July 23-29, 1954. Prof. A. Prudente, 171 rua Benjamin Constante, Sao Paulo, Brazil, President.

INTERNATIONAL CONFERENCE ON THROMBOSIS AND EMBOLISM, Basle, Switzerland, July 20-24, 1954. Dr. W. Merz, Chief Medical Officer, Gynecological Clinic, University of Basle, Basle, Switzerland, Hon. Secretary.

INTERNATIONAL CONGRESS ON CHILD PSYCHIATRY, University of Toronto, Toronto, Ontario, Canada, Aug. 13-14, 1954. Miss Helen Speyer, International Association for Child Psychiatry, 1790 Broadway, New York 19, New York, U. S. A., Executive Officer.

INTERNATIONAL CONGRESS OF CLINICAL PATHOLOGY, Washington, D. C., U. S. A., Sept. 6-10, 1954. Dr. Robert A. Moore, Washington University School of Medicine, St. Louis 10, Missouri, U. S. A., Chairman, Committee on Arrangements.

INTERNATIONAL CONGRESS ON DISEASES OF THE CHEST, Barcelona, Spain, Oct. 4-8, 1954 Mr. Murray Kornfeld, 112 East Chestnut St., Chicago 11, Illinois, Executive Secretary.

INTERNATIONAL CONGRESS ON GROUP PSYCHOTHERAPY, Toronto, Ont., Canada, Aug. 12-14, 1954. Dr. J. L. Moreno, Room 327, 101 Park Ave., New York 17, N. Y., U. S. A., Director of Organizing Committee.

INTERNATIONAL CONGRESS ON GYNECOLOGY AND OBSTETRICS, Geneva, Switzerland, July 26-31, 1954. Dr. H. de Watteville, Maternité Hôpital Cantonal, Geneva, Switzerland, President.

INTERNATIONAL CONGRESS OF HEMATOLOGY, Paris, Sept. 6-11, 1954. Dr. Jean Bernard, 86 rue d'Assas, Paris 6e, France, Secretary.

INTERNATIONAL CONGRESS OF THE HISTORY OF MEDICINE, Rome and Salerno, Italy, Sept. 13-20, 1954. For information write: Segreteria XIV Congresso Internazionale di Storia della Medicina, Instituto di Storia della Medicine, Citta Universitaria, Rome, Italy.

INTERNATIONAL CONGRESS OF HYDROCLIMATISM AND THALASSOTHERAPY, Dubrovnik, Yugoslavia, May 8-16, 1954. Prof. C. Plavsic, Zeleni venac 1, Belgrade, Yugoslavia, Secretary General.

INTERNATIONAL CONGRESS OF INDUSTRIAL MEDICINE, Naples, Italy, Sept. 13-19, 1954. Professor Scipione Caccuri, Director, Institute of Industrial Medicine Policlinico, Naples, Italy, Chairman, Organizing Committee.

INTERNATIONAL CONGRESS OF INTERNAL MEDICINE, Stockholm, Sweden, Sept. 15-18, 1954. Professor Anders Kristenson, Karolinska Sjukhuset, Stockholm 60, Sweden, Secretary-General.

INTERNATIONAL CONGRESS OF INTERNATIONAL COLLEGE OF SURGEONS, São Paulo, Brazil, April 26-May 2, 1954. Dr. Max Thorek, 1516 Lake Shore Drive, Chicago, Illinois, U. S. A., Secretary-General.

INTERNATIONAL CONGRESS ON MENTAL HEALTH, University of Toronto, Toronto, Ontario, Canada, Aug. 14-21, 1954. For information write: Executive Officer, International Congress on Mental Health, 111 St. George St., Toronto, Ontario, Canada.

INTERNATIONAL CONGRESS OF MILITARY MEDICINE AND PHARMACY, Buenos Aires, Argentine, April 21-28, 1954. Direcion General de Sanidad Militar, Pozos 2045, Buenos Aires, Argentine.

INTERNATIONAL CONGRESS OF NUTRITION, Amsterdam, Netherlands, Sept. 14-18, 1954. For information write: Dr. M. van Eekelen, Centraal Instituut voor Voedingsonderzoek T.N.O., 61 Catharynesingel, Utrecht, Netherlands.

INTERNATIONAL CONGRESS OF OPHTHALMOLOGY, University of Montreal and McGill University, Montreal, Canada, Sept. 10-11, 1954, and Waldorf-Astoria, New York, N. Y., U. S. A., Sept. 12-17, 1954. Dr. William L. Benedict, 100 First Avenue Building, Rochester, Minnesota, U. S. A., Secretary-General.

INTERNATIONAL CONGRESS OF ORTHOPEDIC SURGERY AND TRAUMATOLOGY, Berne, Switzerland, Aug. 30-Sept. 3, 1954. For information write: Professor M. Dubois, Isle-Hospital, Berne, Switzerland.

INTERNATIONAL CONGRESS OF PSYCHOLOGY, Montreal, Canada, June 7-12, 1954. For information write: Prof. H. S. Langfeld, International Union of Scientific Psychology, Eno Hall, Princeton University, Princeton, N. J., U. S. A.

INTERNATIONAL CONGRESS FOR PSYCHOTHERAPY, Zurich, Switzerland, July 21-24, 1954. Dr. H. K. Fierz, Theaterstrasse 12, Zurich 1, Switzerland, Secretary General.

INTERNATIONAL CONGRESS OF THE SOCIEDAD DE MÉDICOS INTERNOS, Residentes y Becarios del Instituto Nacional de Cardiologia de Mexico, Acapulco, Mexico, April 21-24, 1954. For information address: Dr. Jorge Soberón Acevedo, Avenida, Cuauhtemoc No. 300, Mexico, D. F., Mexico.

INTERNATIONAL GERONTOLOGICAL CONGRESS, London and Oxford, England, July 12-22, 1954. Prof. R. E. Tunbridge, General Infirmary, Department of Medicine, The University, Leeds, England, President.

INTERNATIONAL POLIOMYELITIS CONGRESS, Rome, Italy, Sept. 6-10, 1954. Mr. Stanley E. Henwood, 120 Broadway, New York 5, N. Y., U. S. A., Executive Secretary.

INTERNATIONAL SOCIETY OF BLOOD TRANSFUSION, Paris, France, Sept. 12-19, 1954. For information write: Colonel Julliard, Société Internationale de Transfusion Sanguine, 53 Boulevard Diderot, Paris 12e, France.

INTERNATIONAL SOCIETY FOR CELL BIOLOGY, Leiden, Netherlands, Sept. 2-9, 1954. Professor Peter J. Gaillard, University of Leiden, Leiden, Netherlands, Secretary.

INTERNATIONAL SOCIETY OF GEOGRAPHICAL PATHOLOGY, Washington, D. C., U. S. A., Sept. 6-10, 1954. Professor Fred C. Roulet, Hebelstrasse 24, Basle, Switzerland, Secretary-General.

IRISH MEDICAL ASSOCIATION, Killarney, Ireland, July 7-10, 1954. Dr. P. J. Delaney, 10, Fitzwilliam Place, Dublin, Ireland, Medical Secretary.

JOURNEES MEDICALES, Paris, France, April 21-25, 1954. For information write: Secretariat of the Journees, 12, rue Pierre-Geofroix, Colombes (Seine) France.

LATIN AMERICAN CONGRESS ON GYNECOLOGY AND OBSTETRICS, Sao Paulo, Brazil, July 10-15, 1954. Prof. Dr. Jairo Ramos, av. Brigaderio Luiz Antonio, 278-8° andar, Sao Paulo, Brazil, Chairman of Organizing Committee of Medical Congresses.

LATIN AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY, Caracas, Venezuela, Feb. 21-25, 1954. Dr. Victorino Marquez Reveron, Centro, Medico, Caracas, Venezuela, Secretary-General.

LATIN AMERICAN CONGRESS OF PHYSICAL MEDICINE, Medellin, Columbia, South America, Feb. 15-20, 1954. Dr. Cassius Lopez de Victoria, 176 East 71st St., New York 21, N. Y., U. S. A., Executive Director.

PAN AMERICAN CONGRESS OF CHILD WELFARE AND PEDIATRICS, Sao Paulo, Brazil, July 15-21, 1954. For information address: Dr. Jairo Ramos, Avenida Brigaderio Luiz Antonio 278-8° andar, Sao Paulo, Brazil.

PAN AMERICAN CONGRESS OF GASTROENTEROLOGY, Sao Paulo, Brazil, July 19-24, 1954. For information address: Dr. Jairo Ramos, Avenida Brigadeiro Luiz Antonio 278-8° andar, Sao Paulo, Brazil.

PAN AMERICAN CONGRESS OF OPHTHALMOLOGY (Interim), São Paulo, Brazil, June 17-21, 1954. Dr. Moacyr E. Alvaro, São Paulo, Brazil, President.

PAN AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY, Mexico, D.F., Mexico, Feb. 28-March 4, 1954. Dr. Maximo Garcia Castañeda, Humboldt 17, Mexico 1, D.F., Mexico, Secretary-General.

PAN AMERICAN CONGRESS OF VETERINARY MEDICINE, Sao Paulo, Brazil, April 3-10, 1954. Dr. Joao Soares Veiga, Rua Pires da Mota 159, Sao Paulo, Brazil, Chairman of Organizing Committee.

PAN-PACIFIC SURGICAL CONGRESS, Honolulu, Hawaii, Oct. 7-18, 1954. Dr. F. J. Pinkerton, Suite 7, Young Bldg., Honolulu 13, Hawaii, Director General.

SECTIONAL MEETING, AMERICAN COLLEGE OF SURGEONS, London, England, May 17-19, 1954. Dr. Michael L. Mason, 40 East Erie St., Chicago 11, Ill., U. S. A., Secretary.

SOUTH AMERICAN CONGRESS OF ANGIOLOGY, Sao Paulo, Brazil, July 1954. For information write: Dr. Rubens Carlos Mayall, Rua Senador Vergueiro 73, Rio de Janeiro, Brazil, S. A.

WORLD CONGRESS OF CARDIOLOGY, Washington, D. C., and Bethesda, Md., U. S. A., Sept. 12-17, 1954. Dr. L. W. Gorham, 44 East 23d St., New York 10, N. Y., U. S. A., Secretary-General.

WORLD CONGRESS OF INTERNATIONAL SOCIETY FOR THE WELFARE OF CRIPPLES, Scheveningen-The Hague, Netherlands, Sept. 13-17, 1954. Secretariat: Miss H. P. Post, Pieter Lastmarkade 37, Amsterdam Z, Netherlands.

WORLD FEDERATION OF OCCUPATIONAL THERAPISTS, Edinburgh, Scotland, August 17, 1954.

WORLD MEDICAL ASSOCIATION, Rome, Italy, Sept. 26-Oct. 2, 1954. Dr. Louis H. Bauer, 345 East 46th St., New York 17, N. Y., U. S. A., Secretary-General.

EXAMINATIONS AND LICENSURE

BOARDS OF MEDICAL EXAMINERS

ALABAMA: *Examination.* Montgomery, June 22-24, 1954. Sec., Dr. D. G. Gill, 537 Dexter Ave., Montgomery.

ARKANSAS:* *Examination.* Little Rock, June 10-11. Sec., Dr. Joe Verser, Harrisburg. *Eclectic.* Little Rock, June 11-12. Sec., Dr. O. L. Atkinson, 2528 Central Ave., Hot Springs National Park.

CALIFORNIA: *Written.* Los Angeles, March 15-18; San Francisco, June 21-24; San Diego, Aug. 23-26; Sacramento, Oct. 18-21. *Oral.* Los Angeles, March 13; San Francisco, June 19; San Diego, Aug. 21; Los Angeles, Nov. 20. *Oral and Clinical Examination for Foreign Medical School Graduates.* Sec., Dr. Louis E. Jones, 1020 N Street, Sacramento.

COLORADO:* *Examination.* Denver, June 8-9. Final date for filing applications is May 8. *Reciprocity.* Denver, April 13. Final date for filing applications is March 13. Exec. Sec., Mrs. Beulah H. Hudgens, 831 Republic Bldg., Denver 2.

CONNECTICUT:* *Examination.* Hartford, March 9-10. Secretary to the Board, Dr. Creighton Barker, 160 St. Ronan St., New Haven. *Homeopathic.* Derby, March 9-10. Sec., Dr. Donald A. Davis, 38 Elizabeth St., Derby.

DISTRICT OF COLUMBIA:* *Examination.* Washington, May 10-11. Deputy Director, Mr. Paul Foley, Department of Occupations and Professions, 1740 Massachusetts Ave., N.W., Washington, D. C.

GEORGIA: *Examination and Reciprocity.* Atlanta and Augusta, June. Sec., Mr. R. C. Coleman, 111 State Capitol, Atlanta 3.

GUAM: The Commission on Licensure will meet whenever a candidate appears or submits his credentials. Director of Medical Services, Guam Memorial Hospital, Agana

ILLINOIS: *Examination and Reciprocity.* Chicago, Jan. 26-28; April 6-8; June 22-24, and Oct. 5-7. Supt. of Registration, Mr. Fredric B. Selcke, Capitol Bldg., Springfield.

INDIANA: *Examination.* Indianapolis, June 16-18. Exec. Sec., Miss Ruth V. Kirk, 538 K of P Bldg., Indianapolis.

KANSAS: *Examination and Reciprocity.* Kansas City, June 9-10. Sec., Dr. O. W. Davidson, 872 New Brotherhood Bldg., Kansas City.

MAINE: *Examination and Reciprocity.* Portland, March 9-10. Sec., Dr. Adam P. Leighton, 192 State St., Portland.

MARYLAND: *Examination.* Baltimore, June 15-18. Sec., Dr. E. H. Kloman, 1215 Cathedral St., Baltimore 1.

MICHIGAN:* *Examination.* Detroit and Ann Arbor, June 1954. Sec., Dr. J. Earl McIntyre, 202-4 Hollister Bldg., Lansing 8.

MISSISSIPPI: *Examination and Reciproctiy.* Jackson, June. Asst. Sec., Dr. R. N. Whitfield, Old Capitol, Jackson 113.

MISSOURI: *Examination.* Jefferson City, Feb. 18-19. *Reciprocity.* Jefferson City, Feb. 8. Exec. Sec., Mr. John A. Hailey, P. O. Box 4, Jefferson City.

MONTANA: *Reciprocity.* Helena, April 5. *Examination.* Helena, April 6-7. Sec., Dr. S. A. Cooney, 214 Power Block, Helena.

NEBRASKA:* *Examination.* Omaha, June 1954. Director, Mr. Husted K. Watson, 1009 State Capitol Bldg., Lincoln.

NEW HAMPSHIRE: *Examination and Reciprocity.* Concord, March 10. Sec., Dr. John S. Wheeler, 107 State House, Concord.

NEW JERSEY: *Examination,* Trenton, June 15-18. Sec., Dr. E. S. Hallinger, 28 W. State St., Trenton.

NEW MEXICO:* *Examination and Reciprocity.* Santa Fe, April 12-13. Sec., Dr. R. C. Derbyshire, 227 E. Palace Ave., Santa Fe.

NEW YORK: *Examination.* Albany, Buffalo, Syracuse and New York City, Feb. 16-19. Sec., Dr. Stiles D. Ezell, 23 S. Pearl St., Albany.

OHIO: *Examination.* Columbus, June 14-16. *Reciprocity.* Columbus, April 13. Sec., Dr. H. M. Platter, 21 W. Broad St., Columbus.

OKLAHOMA:* *Examination.* Oklahoma City, June 9-10. Sec., Dr. Clinton Gallaher, 813 Braniff Bldg., Oklahoma City.

PENNSYLVANIA: *Examination.* Philadelphia, January 1954. Acting Secretary, Mrs. Margaret G. Steiner, Box 911, Harrisburg.

SOUTH CAROLINA: *Reciprocity.* Columbia, Feb. 1. Sec., Mr. N. B. Heyward, 1329 Blanding St., Columbia.

SOUTH DAKOTA:* *Examination.* Sioux Falls, Jan. 19-20, 1954. Reciprocity can be obtained at any time between meetings of board. Executive Secretary, Mr. John C. Foster, 300 First National Bank Bldg., Sioux Falls.

TENNESSEE:* *Examination.* Memphis, March 24-25. Sec., Dr. H. W. Qualls, 1635 Exchange Bldg., Memphis.

TEXAS: *Examination and Reciprocity.* Fort Worth, June 21-23. Sec., Dr. M. H. Crabb, 1714 Medical Arts Bldg., Ft. Worth 2.

UTAH: *Reciprocity.* Salt Lake City, Feb. 16. Asst. Director, Mr. Frank E. Lees, 314 State Capitol Bldg., Salt Lake City 1.

VERMONT: *Examination.* Burlington, Jan. 28-30. Sec., Dr. F. J. Lawliss, Richford.

VIRGINIA: *Examination and Reciprocity.* Richmond, June 16. Address: Virginia Board of Medical Examiners, 631 First St., S.W., Roanoke.

WYOMING: *Oral Examination and Reciprocity.* Cheyenne, Feb. 1. Sec., Dr. Franklin D. Yoder, State Office Bldg., Cheyenne.

BOARDS OF EXAMINERS IN THE BASIC SCIENCES

ALASKA: On application. Juneau or other towns in Territory as decided by Board. *Reciprocity.* On application. Sec., Dr. C. Earl Albrecht, Box 1931, Juneau.

ARIZONA: *Examination.* Tucson, March 16. Sec., Mr. Herbert D. Rhodes, University of Arizona, Tucson.

COLORADO: *Examination.* Denver, March 3-4. Sec., Dr. Esther B. Starks, 1459 Ogden St., Denver 18.

CONNECTICUT: *Examination.* New Haven, Feb. 13. Address: State Board of Healing Arts, 52 Whitney Ave., New Haven 10.

FLORIDA: *Examination.* Gainesville, June. Sec., Mr. M. W. Emmel, University of Florida, Box 340, Gainesville.

MICHIGAN: *Examination.* Detroit and Ann Arbor, Feb. 12-13. Sec., Mrs. Anne Baker, 410 W. Allegan St., Lansing 15.

NEBRASKA: *Examination.* Omaha, May 4-5. Director, Mr. Husted K. Watson, 1009 State Capitol Bldg., Lincoln 9.

OKLAHOMA: *Examination.* Oklahoma City, April 16-17. Sec., Dr. Clinton Gallaher, 813 Braniff Bldg., Oklahoma City.

OREGON: *Examination.* Portland, March 6, June 5, Sept. 11 and Dec. 4. Sec., Mr. Charles D. Byrne, State Board of Higher Education, Eugene.

RHODE ISLAND: *Examination.* Providence, Feb. 10. Administrator, Division of Professional Regulation, Mr. Thomas B. Casey, 366 State Office Bldg., Providence.

TEXAS: *Examination.* April 1954. Location determined by number of applicants from different sections of the state. Sec., Brother Raphael Wilson, C.S.C., 407 Perry-Brooke Bldg., Austin.

WISCONSIN: *Examination.* Madison, April 3 and June 5. Final date for filing application is March 26 and May 28. Sec., Dr. W. H. Barber, 621 Ransom St., Ripon.

*Basic Science Certificate required.

DEATHS

Binion, Richard ⊕ Milledgeville, Ga.; born in Sparta, Ga., July 19, 1892; University of Maryland School of Medicine, Baltimore, 1915; also a graduate in pharmacy; member of the Southeastern Surgical Congress, of which he was formerly a member of the council, and the American Psychiatric Association; fellow of the American College of Surgeons; past president of the Baldwin County Medical Society; past president of the Georgia Railroad Surgeons Association; served during World War I; formerly chairman of the Baldwin County Board of Health and vice-chairman of the state board of public welfare; at one time physician for the Georgia State College for Women, Georgia Military College, and the Milledgeville State Training Schools for Boys; formerly member of the board of governors of the Milledgeville State Hospital; retired owner of the Richard Binion Clinic Hospital; for many years associated with the Allen's Invalid Home; died Oct. 22, aged 62, of arteriosclerotic heart disease.

Golden, Waldo Emerson ⊕ Commander, U. S. Navy, retired, Berkeley, Calif.; born at Flora, Ill., June 9, 1893; University of Illinois College of Medicine, Chicago, 1917; entered the Medical Corps, U. S. Navy, as a lieutenant, junior grade; promoted to lieutenant in 1920 and to lieutenant commander in 1925; placed on the retired list of officers of the Navy July 1, 1941, and immediately recalled to active duty; was promoted to commander in the medical corps, Jan. 3, 1942, and released from active duty July 1, 1945; served during World Wars I and II; during his 28 years of naval service, served in the U. S. ships Ramapo and Chaumont, at the Naval Medical School, Washington, D. C., the Naval Training Station and Naval Hospital, Great Lakes, Ill., the Naval Hospital, Mare Island, Calif., the Recruiting Station, San Francisco, and the Naval Supply Depot in Oakland, Calif.; died Nov. 27, aged 60.

Graves, Marvin Lee ⊕ Houston, Texas; born in Bosqueville, Texas, March 26, 1867; Bellevue Hospital Medical College, New York, 1891; professor emeritus of internal medicine at the University of Texas School of Medicine, Galveston; fellow of the American College of Physicians; member of the House of Delegates of the American Medical Association from 1914 to 1919; past president of the State Medical Association of Texas; for many years consulting physician at the John Sealy Hospital in Galveston; from 1898 to 1905 superintendent of the Southwestern Insane Asylum in San Antonio; in 1918 received the L.L.D. degree from Southern Methodist University in Dallas; formerly medical director, American General Life Insurance Company; died in Methodist Hospital Nov. 19, aged 86, of encephalomalacia.

Griffitts, Thomas Houston Dow ⊕ medical director, U. S. Public Health Service, retired, Fort Lauderdale, Fla.; born Oct. 26, 1878; Chattanooga (Tenn.) Medical College, 1903; past president of the Florida Public Health Association; in charge of the Henry R. Carter Memorial Laboratory of the U. S. Public Health Service, Savannah, Ga., when it was opened in 1937, and remained until 1939; twice served the city of Savannah as acting health officer; formery chief quarantine officer of Puerto Rico and the Virgin Islands, with headquarters in San Juan; retired from the U. S. Public Health Service Nov. 1, 1942, after 30 years as a medical officer; formerly director of the Dade County Health Unit, Miami; died in the U. S. Marine Hospital, Baltimore, Oct. 26, aged 75.

Acker, Paul Jerome Moeris ⊕ Mobile, Ala.; Medical College of Alabama, Mobile, 1892; fellow of the American College of Surgeons; formerly on the faculty of his alma mater; for many years county health officer; at one time associated with the U. S. Public Health Service; served as chief of staff at the City Hospital; died Nov. 28, aged 82, of carcinoma of the colon.

⊕ Indicates Member of the American Medical Association.

Anderson, Joseph R., Sumrall, Miss.; Memphis (Tenn.) Hospital Medical College, 1902; a member and formerly president of the school board; one of the founders of the Bank of Sumrall and served on the board of directors; died in the Methodist Hospital, Hattiesburg, Nov. 11, aged 77.

Annunziata, Augustine James ⊕ New York City; University of Buffalo School of Medicine, 1924; served during World War II; on the staff of the Mother Cabrini Hospital; died Nov. 19, aged 54, of heart failure.

Ashford, Hartwell T., Clinton, Miss.; Hospital College of Medicine, Louisville, 1903; served as physician for Mississippi College; died Dec. 15, aged 75.

Aubuchon, William E., Frankclay, Mo.; American Medical College, St. Louis, 1909; died Oct. 28, aged 74.

Baker, Robert Whitney ⊕ Chicago; University of Illinois College of Medicine, Chicago, 1945; affiliated with the Veterans Administration Hospital in Livermore, Calif.; died in Fort Miley Hospital in San Francisco Nov. 4, aged 33, of bacterial endocarditis.

Bauersfeld, Emil George ⊕ Bethesda, Md.; Baltimore Medical College, 1911; died Dec. 13, aged 64.

Baumann, Harry Floyd, Meridian, Pa.; University of Pittsburgh School of Medicine, 1913; died recently, aged 66.

Bayer, Ira Eugene Jr., ⊕ Baltimore; University of Maryland School of Medicine, Baltimore, 1934; died Oct. 24, aged 47.

Baylor, John Ward ⊕ Bethesda, Md.; Johns Hopkins University School of Medicine, Baltimore, 1918; formerly associate professor of laryngology and otology at Johns Hopkins University School of Medicine in Baltimore, where he was affiliated with the Woman's Hospital, Church Home and Infirmary, and the Union Memorial Hospital; died Nov. 25, aged 60, of cardiovascular disease.

Bearer, Albert John, New Kensington, Pa.; Cincinnati College of Medicine and Surgery, 1897; died in the Citizens General Hospital Sept. 4, aged 82.

Bennett, Edward, Monroe, Ore.; California Medical College, San Francisco, 1895; St. Louis College of Physicians and Surgeons, 1900; served on the staff of the Good Samaritan Hospital in Corvallis; died Nov. 27, aged 79, of angina pectoris.

Benson, Andrew Ludwig ⊕ Philipsburg, Pa.; Jefferson Medical College of Philadelphia, 1915; member of the Radiological Society of North America; owner and chief surgeon of the Benson Sanatorium; died Dec. 3, aged 61.

Bogen, Eugene Feilen, Rochester, N. Y.; Chicago College of Medicine and Surgery, 1913; University of Kharkov Faculty of Medicine, Russia, 1918; specialist certified by the American Board of Psychiatry and Neurology; member of the American Psychiatric Association; served during World War II, and on the staffs of various Veterans Administration hospitals; died Dec. 17, aged 67.

Boggs, Lloyd Kennedy ⊕ Birmingham, Ala.; University of Georgia Medical Department, Augusta, 1924; died in the Jefferson Hospital Oct. 16, aged 56, of melanoma with metastasis.

Bregel, Fred Louis ⊕ St. Paul; University of Minnesota Medical School, Minneapolis, 1918; served on the rating board of the Veterans Administration at Fort Snelling, Minn., and St. James (Minn.) Hospital; died in St. Joseph Hospital Nov. 13, aged 64, of cerebral thrombosis.

Bright, Fred L., Battle Creek, Mich.; Michigan College of Medicine and Surgery, Detroit, 1904; died in the Lelia Y. Post Montgomery Hospital Oct. 15, aged 79, of cardiac insufficiency.

Brooks, Thomas Gibson ⊕ Aiken, S. C.; University of Georgia Medical Department, Augusta, 1924; served during World War II; fellow of the American College of Surgeons; on the staff of Aiken County Hospital; died in the University Hospital, Augusta, Ga., Oct. 27, aged 53, of rheumatoid arthritis.

Brown, Crawford MacDowell ⊕ Rochester, N. Y.; University of Pittsburgh School of Medicine, 1931; served during World War II; affiliated with Highland Hospital; died Dec. 3, aged 50.

Carrington, Samuel Macon ⊕ Oxford, N. C.; Rush Medical College, Chicago, 1931; served during World War II; died in Durham Oct. 29, aged 49, of cerebral hemorrhage and leukemia.

Conner, Leonidas J., Kirksville, Mo.; Marion-Sims College of Medicine, St. Louis, 1892; died in the Grim Smith Memorial Hospital Oct. 8, aged 84, of coronary occlusion.

Cooper, John Hightower ⊕ Commerce, Ga.; Atlanta College of Physicians and Surgeons, 1913; member of the Florida Medical Association; served during World War I; practiced in St. Petersburg, Fla.; formerly affiliated with Wesley Long Hospital in Greensboro, N. C.; died in the Commerce Hospital Nov. 14, aged 66, of coronary occlusion and cirrhosis of the liver.

Coughlin, Francis Joseph ⊕ Arlington, N. J.; McGill University Faculty of Medicine, Montreal, Canada, 1917; died Nov. 28, aged 58, of cerebral hemorrhage, diabetes mellitus, and hypertension.

Cullison, Robert M., ⊕ Winston Salem, N. C.; State University of Iowa College of Medicine, Iowa City, 1917; member of the Iowa State Medical Society; served during World Wars I and II; chief medical officer of the regional office of the Veterans Administration in North Carolina; died Nov. 4, aged 62, of coronary thrombosis and arteriosclerosis.

Dalton, William Nicholson ⊕ Winston-Salem, N. C.; North Carolina Medical College, Davidson, 1904; died in the City Memorial Hospital Oct. 23, aged 72, of coronary occlusion and arteriosclerosis.

Donovan, Ophius Poston, Bismarck, Ill.; St. Louis College of Physicians and Surgeons, 1903; died in the Lake View Hospital, Danville, recently, aged 74, of heart and kidney disease.

Duval, Joseph Berwick ⊕ Houma, La.; Medical Department of Tulane University of Louisiana, New Orleans, 1903; died Dec. 8, aged 77.

Earl, Franklin Daniels ⊕ Ogdensburg, N. Y.; Bellevue Hospital Medical College, New York, 1895; an Associate Fellow of the American Medical Association; formerly school physician and district health officer; affiliated with A. Barton Hepburn Hospital; died Nov. 23, aged 82, of cerebral hemorrhage and arteriosclerosis.

Farrar, Charles O., ⊕ Nettleton, Miss.; Memphis (Tenn.) Hospital Medical College, 1901; on the staff of North Mississippi Community Hospital in Tupelo; died Dec. 4, aged 81, of dilatation of the heart.

Fernald, Arthur May ⊕ Concord, N. H.; Baltimore Medical College, 1898; died Oct. 31, aged 82, of coronary thrombosis and hypertension.

Feuerstein, Harry B., ⊕ St. Albans, N. Y.; University and Bellevue Hospital Medical College, New York, 1917; served during World War I; affiliated with the University Hospital in New York City, where he died Dec. 1, aged 59, of coronary thrombosis.

Frink, Raymond Peter, Redfield, S. D.; Northwestern University Medical School, Chicago, 1903; served during World War I; formerly physician in the Indian Service; died in the State School and Home for Feebleminded Oct. 25, aged 76, of chronic heart disease and glomerulonephritis.

Fuller, Melvin Francis ⊕ Aberdeen, Wash.; University of Oregon Medical School, Portland, 1933; served during World War II; fellow of the American College of Surgeons; on the staff of St. Joseph's Hospital, where he died Nov. 13, aged 49, of adenocarcinoma.

Goodale, Banks H. ⊕ Jacksonville, Fla.; Southern College of Medicine and Surgery, Atlanta, 1912; died in St. Luke's Hospital Oct. 8, aged 65, of bronchopneumonia.

Harris, Harold Shelton ⊕ Troy, Mo.; Washington University School of Medicine, St. Louis, 1925; served during World War II; died Oct. 23, aged 56.

Harris, Lurty Noel, Wheeling, W. Va.; University of Maryland School of Medicine, Baltimore, 1897; died in the Ohio Valley Hospital Sept. 17, aged 79, of arteriosclerosis.

Harris, Monroe, Bloomington, Ill.; Northwestern University Medical School, Chicago, 1909; practiced in Benton; died in the Mennonite Hospital Sept. 13, aged 83, of cerebral hemorrhage.

Hawkins, Martin Cassetty Sr., ⊕ Parkdale, Ark.; University of Louisville, Ky., Medical Department, 1893; affiliated with Lake Village Infirmary in Lake Village, and Hawkins Clinic Hospital in Searcy; died Nov. 28, aged 84, of pneumonia.

Henderson, Harold Burrows ⊕ Denver; Northwestern University Medical School, Chicago, 1923; formerly assistant professor of obstetrics and gynecology at University of Colorado School of Medicine; fellow of the American College of Surgeons; served during World War I; past chief of staff of Presbyterian and Booth Memorial hospitals; died Sept. 24, aged 64, of coronary thrombosis.

Henry, James R., Kansas City, Mo.; Kansas City Hahnemann Medical College, 1907; died in the Research Hospital Oct. 13, aged 69.

Hill, Robert Burns ⊕ Beverly Hills, Calif.; University of Missouri School of Medicine, Columbia, 1909; fellow of the American College of Physicians; affiliated with California Hospital; died Nov. 12, aged 67, of carcinoma of the larynx.

Hoffman, Burton Louis, Chicago; University of Chicago, the School of Medicine, 1941; assistant professor of neurosurgery at his alma mater; lieutenant commander in the U. S. Navy during World War II; resigned from the Navy on May 30, 1947; formerly a resident in neurosurgery at the University of Chicago Clinics; died in Albert Merritt Billings Hospital Dec. 2, aged 37, of adrenal cortical carcinoma with pulmonary and hepatic metastases.

Hunter, Charles Elverton ⊕ Mansfield, Ohio; Starling Medical College, Columbus, 1902; on the courtesy staff at the Mansfield General Hospital, where he died Nov. 22, aged 76, of leukemia.

Hunter, John Alexander ⊕ Dover, N. H.; University of Vermont College of Medicine, Burlington, 1911; died Oct. 2, aged 66, of cerebral hemorrhage and arteriosclerosis.

Kisor, Frank Hays, Winter Haven, Fla.; State University of Iowa College of Medicine, Iowa City, 1895; died in Lake Alfred Oct. 24, aged 88, of uremia, nephrosclerosis, and arteriosclerosis.

Krejsa, Oldrich, Cedar Rapids, Iowa; State University of Iowa College of Medicine, Iowa City, 1901; died Aug. 8, aged 78, of myocardial infarction.

Landreth, John J. ⊕ Bruce, Miss.; Memphis (Tenn.) Hospital Medical College, 1898; died Nov. 13, aged 85, of a heart attack.

Langdale, Charles Andrew ⊕ Coral Gables, Fla.; Medical College of Ohio, Cincinnati, 1903; served on the faculty of the University of Cincinnati College of Medicine; fellow of the American College of Surgeons; formerly practiced in Cincinnati, where he was affiliated with Good Samaritan and Christ hospitals; died in Coral Gables Hospital Oct. 26, aged 79, of arteriosclerotic heart disease and hypertension.

Lange, Carl Wilhelm, Albany, N. Y.; Hessische Ludwigs-Universität Medizinische Fakultät, Giessen, Hesse, Germany, 1907; died Oct. 26, aged 70.

Leake, William Samuel Jr., ⊕ Lafayette, La.; Tulane University of Louisiana School of Medicine, New Orleans, 1937; member of the Tennessee State Medical Association; served during World War II; died Nov. 10, aged 43, of coronary occlusion.

Lewis, Benjamin Jefferson, Montgomery, Ala.; Medical College of Alabama, Mobile, 1899; died Nov. 20, aged 79, of injuries received in an automobile accident.

Lieberman, William, Jamaica, N. Y.; New York Homeopathic Medical College and Flower Hospital in New York, 1917; died suddenly Aug. 10, aged 58.

Loomis, Edgar Webb ✠ Dallas, Texas; Southwestern University Medical College, Dallas, 1910; specialist certified by the American Board of Pediatrics; member of the American Academy of Pediatrics; formerly city health officer; served in France during World War I; for two years a medical missionary in Alaska for the Episcopal Church; affiliated with Methodist, Parkland, and St. Paul's hospitals; died Nov. 18, aged 67.

Lovelady, William Henry ✠ Hartselle, Ala.; Medical College of Alabama, Mobile, 1897; died Nov. 29, aged 84.

McAfee, Frederick W., ✠ Detroit; University of Michigan Homeopathic Medical School, Ann Arbor, 1904; served during World War I; died Nov. 28, aged 75, of myocardial infarction.

McBratney, Emmett William ✠ Tucson, Ariz.; Washington University School of Medicine, St. Louis, 1903; an Associate Fellow of the American Medical Association; member of the Missouri State Medical Association; at one time practiced in St. Louis; died in Columbia, Mo., Oct. 24, aged 78.

McGehee, Henry Thomas ✠ Birmingham, Ala.; Medical College of Alabama, Mobile, 1904; died in the Lloyd Noland Hospital, Fairfield, Oct. 18, aged 78, of myocardial infarction.

McIver, Bert Andrew ✠ Lowry, Minn.; University of Minnesota Medical School, Minneapolis, 1929; died in the Glenwood (Minn.) Community Hospital, Oct. 31, aged 56, of acute bronchopneumonia.

Mason, William C., Murphy, N. C.; Tennessee Medical College, Knoxville, 1904; died Oct. 23, aged 81, of cerebrovascular accident and arteriosclerosis.

Matthews, William Thomas, New Vienna, Ohio; Medical College of Ohio, Cincinnati, 1901; member of the board of directors of the New Vienna Bank; died in the White Cross Hospital, Columbus, Nov. 30, aged 82, of myocarditis and pulmonary congestion following a hip fracture received in a fall.

Menninger, Charles Fredric ✠ Topeka, Kan.; the Hahnemann Medical College and Hospital, Chicago, 1889; past president of the Shawnee County Medical Society; member of the Endocrine Society, American Psychiatric Association, and the Central Neuropsychiatric Association; served as president of the Topeka Board of Health; founder of the Menninger Clinic and chairman of the board of trustees of the Menninger Foundation; died Nov. 28, aged 91, of cerebral thrombosis.

Mills, Edmund Dumas ✠ Beaumont, Texas; University of Texas School of Medicine, Galveston, 1916; died recently, aged 61, of coronary occlusion.

Moylan, David John, Bala-Cynwyd, Pa.; University of Pennsylvania Department of Medicine, Philadelphia, 1906; served on the staffs of Wills Eye Hospital, St. Vincent's Hospital for Women and Children, and St. Joseph's Hospital; died Dec. 3, aged 80, of coronary occlusion.

Mullen, Robert Ambrose ✠ Tampa, Fla.; Marquette University School of Medicine, Milwaukee, 1926; member of the American Society of Anesthesiologists and the State Medical Society of Wisconsin; served during World War II; died in St. Joseph's Hospital Nov. 5, aged 53, of coronary thrombosis.

Mumford, Samuel Howard, New Bern, N. C.; Howard University College of Medicine, Washington, D. C., 1926; died Oct. 23, aged 56, of hypertensive cardiovascular disease.

Nemon, Leon ✠ Portland, Maine; Thüringische Landesuniversität Medizinische Fakultät, Jena, Thuringia, Germany, 1928; died Oct. 4, aged 51, of coronary occlusion.

Partlow, Kenneth Lawrence ✠ Olympia, Wash.; Rush Medical College, Chicago, 1914; formerly affiliated with the U. S. Public Health Service; president of the Washington State Medical Association 1950-1951; past president of the Thurston-Mason Counties Medical Society; fellow of the American College of Surgeons; on the staff of St. Peter's Hospital; died Oct. 14, aged 66, of carcinoma of the prostate.

Phelps, John Quincy, Cheyenne, Wyo.; Kentucky School of Medicine, Louisville, 1888; died in the Memorial Hospital Oct. 16, aged 82, of cerebral thrombosis.

Pisciotta, Joseph, Brooklyn; New York Homeopathic Medical College and Hospital, New York, 1914; during World War II, for three years, a member of the board of examiners for a Selective Service Board in Bay Ridge; served on the staffs of St. Mary's and Norwegian Lutheran Deaconesses' Home and Hospital, where he died Nov. 19, aged 66, of pneumonia and acute heart disease.

Prescott, Henry Frederick ✠ Cressona, Pa.; Hahnemann Medical College and Hospital of Philadelphia, 1929; served during World War II and in the Korean conflict; school director and member of the board of health; affiliated with Pottsville and Lemos B. Warne Hospital and Clinic in Pottsville; an associate staff member, Good Samaritan Hospital in Lebanon, where he died Oct. 18, aged 51, of injuries received in an automobile accident.

Roberts, David Allen ✠ Swannanoa, N. C.; University of Oklahoma School of Medicine, Oklahoma City, 1932; served during World War II; affiliated with Veterans Administration Hospital in Oteen, where he died Nov. 25, aged 50, of coronary thrombosis.

Russell, Thomas, Grand Rapids, Minn.; University of Toronto Faculty of Medicine, Toronto, Canada, 1890; died Oct. 15, aged 92.

Schoenfeld, Siegfried M., ✠ New Hyde Park, N. Y.; Medizinische Fakultät der Universität, Vienna, Austria, 1921; member of the American College of Chest Physicians and the American Trudeau Society; on the staff of Terrace Heights Hospital in Hollis; died Nov. 16, aged 57, of acute cardiac failure.

Scott, Harry Alexander ✠ Lincoln, Neb.; University of Buffalo School of Medicine, 1917; specialist certified by the American Board of Radiology; member of the Radiological Society of North America and the American College of Radiology; manager, Veterans Administration Hospital, where he died Nov. 12, aged 63, of hypertensive cardiovascular disease.

Shatto, Arthur Blair ✠ St. Petersburg, Fla.; College of Physicians and Surgeons, Baltimore, 1895; member of the Medical Society of the State of Pennsylvania; died in the Mound Park Hospital, Oct. 11, aged 82, of arteriosclerotic heart disease.

Sheets, Raymond F., Carthage, Ill.; Keokuk (Ia.) Medical College, College of Physicians and Surgeons, 1908; served as district health officer, and as county physician; on the staff of the Memorial Hospital; died suddenly Nov. 25, aged 74, of acute coronary thrombosis.

Spiro, Harry, San Francisco; Cooper Medical College, San Francisco, 1904; at one time on the faculty of Stanford University School of Medicine; served during World War I; on the staff of St. Mary's Hospital; died Nov. 18, aged 78, of heart disease.

DIED WHILE IN MILITARY SERVICE

Meister, Adam George ✠ Sarasota, Fla.; born in Orlando, Fla., Aug. 6, 1924; College of Medical Evangelists, Loma Linda and Los Angeles, 1948; certified by the National Board of Medical Examiners; interned at James M. Jackson Memorial Hospital in Miami, where he later served a residency; in 1952 secretary-treasurer of the Florida chapter of the College of Medical Evangelists Alumni Association; on the staff of the Sarasota Municipal Hospital, serving as secretary of the obstetric section during his last year there; called to active duty as a first lieutenant with the Army in October, 1952; joined the paratroopers the following December, assigned to the 82nd Airborne Division as a regimental surgeon, Fort Bragg, N. C., where he died Nov. 17, aged 29, after an aircraft accident.

GOVERNMENT SERVICES

ARMY

Civilian Physicians Win Army Awards.—The Certificate of Appreciation of the Department of the Army was awarded by Major Gen. George E. Armstrong, Surgeon-General, at ceremonies Nov. 20, to Dr. G. Robert Coatney, of the laboratory of tropical diseases, National Microbiological Institute, U. S. Public Health Service, and Dr. Ralph Jones Jr., assistant professor of research medicine, University of Pennsylvania Medical School, Philadelphia, for research in connection with the development and testing of primaquine, an antimalarial drug.

Dr. Coatney was awarded the certificate for outstanding patriotic civilian service to the Army from January, 1951, to June, 1953, when "he was in charge of malaria chemotherapy studies undertaken by the Surgeon-General to explore the curative effectiveness of several 8-aminoquinolines in prisoner volunteers at Atlanta, Georgia, and in Korean returnees at Fort Benning, and of other tests initiated to determine the antimalarial properties of daraprim," another antimalarial drug. Dr. Jones was awarded the certificate for outstanding patriotic civilian service to the Army from June, 1951, to April, 1953, when "he was responsible for supervising the clinical testing of primaquine as a curative drug for Korean vivax malaria in Army hospitals at Fort Knox, Fort Meade, Camp Breckinridge, Fort Dix, and in the Naval Hospital at the Marine Base at Camp LeJeune." The citation continues, "The efficiency and speed with which primaquine was evaluated on a large scale in its field trials were the direct result of Dr. Jones' efforts in organizing and correlating activities and investigations at the military medical installations concerned."

PUBLIC HEALTH SERVICE

Medical Social Workers.—There are about 3,825 persons actively engaged in medical social work in the U. S. today, according to "Health Manpower Sourcebook, Section III: Medical Social Workers" just released by the Public Health Service. It is said that there are three times as many positions open as there are persons to fill them and that 800 to 1,000 graduates a year will be needed to fill the vacancies. The sourcebook presents, for the first time, a comprehensive study of the employment, educational background, and personal characteristics of medical social workers. The largest number of medical social workers (about 41%) are located in the northeast section of the country. Three-fourths of the total are employed in hospitals and clinics, and the two next largest groups work with the physically handicapped and other special groups. Most medical social workers are women (93%), although the number of male graduates in social work courses is increasing.

Copies of this book are available from the Superintendent of Documents, Government Printing Office, Washington 25, D. C., at 40 cents each (Public Health Service Publication No. 263, Section 3).

Airlines Sanitary Guide.—The first published sanitary guide written expressly for airlines has just been published by the Public Health Service. It is intended as a guide to the air transport industry and for use by the Public Health Service and cooperating state health departments in their periodic inspections of aircraft and servicing facilities. Public law 410 of the 78th Congress authorizes the Public Health Service to prepare regulations for the prevention of the transmission or spread of communicable disease across state lines or into the United States from foreign countries. Mr. Leonard M. Board, chief, division of sanitation, said, "It is hoped that designers, builders, and operators of commercial aircraft will use this handbook as a guide for 'building in' features which will permit sanitary operation."

The airline and catering industries cooperated with the Public Health Service in the preparation of this material. The handbook deals with the review of construction plans covering sanitary facilities both for aircraft and for terminal facilities, sanitary equipment and practices at catering points, sanitary food and beverage service, ice-handling, refuse management on aircraft, sanitary control of drinking water, disposal of wastes from aircraft, and other items of sanitation. The publication is for sale by the Superintendent of Documents, Government Printing Office, Washington 25, D. C.

MISCELLANEOUS

Science Foundation Appoints Committee on Government-University Relationships.—The National Science Foundation, Washington, D. C., has appointed an advisory committee to consider the effects of government support to colleges and universities on their research and teaching functions. The appointment of the committee was authorized by the National Science Board, which selected its own chairman, Chester I. Barnard, to be the chairman of the committee. The other members are:

Arthur S. Adams, president, American Council on Education.
Vannevar Bush, president, Carnegie Institution of Washington.
James S. Coles, president, Bowdoin College.
Harold W. Dodds, president, Princeton University.
Conrad A Elvehjem, dean of the Graduate School, University of Wisconsin.
T. Keith Glennan, president, Case Institute of Technology.
Virgil M. Hancher, president, State University of Iowa.
William V. Houston, president, Rice Institute.
Clark Kerr, chancellor, University of California, Berkeley.
C. N. H. Long, professor of physiology, Yale University.
Don Price, associate director, the Ford Foundation.
Julius A. Stratton, Provost, Massachusetts Institute of Technology.

During World War II and the postwar emergency period many millions of dollars have been spent by government agencies for services in technological developments in universities and colleges. Relatively few millions of dollars have been provided for research and education in the sciences, although in many instances these institutions are uniquely fitted to carry on such activities. Statistics compiled by the foundation indicate that during the year ending June 30, 1952, educational institutions received almost 300 million dollars from federal agencies for development and research. Approximately 136 million dollars (46%) was spent by instructional departments. Of the rest, about 3 million dollars was spent by affiliated research organizations, $12,500,000 by agricultural experiment stations, and $143,500,000 by research centers, which were administered by educational institutions for specific federal agencies but organizationally segregated from the normal activities of the institutions. According to the foundation a total of 225 educational institutions received some federal support for development and research in 1951-1952. These included 86 universities, 95 liberal arts colleges, 41 professional-technical schools, and 3 other educational institutions.

FOREIGN LETTERS

CHILE

Artificial Hibernation.—Dr. Eduardo Escalona Sylvester in the *Bulletin of the Vina del Mar Hospital* (July) reports five cases of artificial pharmacological hibernation during operation. A subtotal gastrectomy for peptic ulcer was performed on two of the patients, and the other three were operated on for extensive cancer of the esophagus. The night before the operation 25 mg. of Phenergan (10-[2-dimethylamino-1-propyl] phenothiazine) with 0.1 gm. of phenobarbital were injected. On the day of the operation, two hours before the anesthesia was begun, 25 mg. of Phenergan was given intramuscularly and one hour later a modification of Laborit's classic "cocktail" was given intravenously. The duration of the administration of the "cocktail" was about one hour, after which period the author used 0.35 to 0.4 gm. of thiopental to produce anesthesia; 80 units of Intocostrin (an extract of Chondodendren tomentosum) and 40 mg. of gallamine (Flaxidil) were given in order to obtain a curare-like effect. Anesthesia was then continued by means of cyclopropane and ether. Blood transfusion was given in the usual manner. The patient's blood pressure usually fell slowly to between 50 and 90 mm. Hg, his pulse rate ranged from 100 to 120, and his skin was dry, pink, and warm during the operation. The postoperative course of these patients was smoother than that of patients of the same type operated on without hibernation. Their rectal temperature was maintained at 93.2 F for 20 hours by the administration of Diparcol (10-[2-dimethylaminoethyl] phenothiazine). The pulse rate, blood pressure, and general condition of the patient were always good. His breathing was deep and quiet; consciousness was not regained till several hours after the operation. In no case was it found necessary to administer analgesics to control pain.

Environmental Study of Normal and Delinquent Boys.—Dr. Alberto Robinson and Miss O. Hinojosa studied the environment of 56 boys, 40 of whom were between 7 and 12 and 16 of whom were between 13 and 17 years of age. A study of the father and/or mother or guardian was also made. Alcoholism was present in about the same percentage of the fathers of the normal and the delinquent boys. Delinquency was overwhelmingly more frequent among the parents of the delinquent children. Neuropsychiatric disturbances were about equally distributed among the parents of both groups. Birth was normal in 52 of the total group. The psychomotor development of the child was difficult to evaluate, but seemed to be abnormal in only 12. The psychological importance of breast feeding in the prevention of frustrations, anxieties, and neurotic complexes was confirmed. Among the delinquents artificial feeding was much more frequent than among the normal boys, most of whom had been breast fed. Of the 39 children who were legitimate, 21 were normal, but, of the 17 who were illegitimate, only 7 were normal. Of 15 who were reared in institutions, 12 were delinquent. In the neighborhood from which the delinquent boys came there were more saloons than in the environment of the control group. Absence from school was more frequent among the delinquent than among the control group. The relation between the parents and the child was given special consideration. An unfavorable attitude of either the son or one of the parents was almost always found in the delinquent group. In some cases this amounted to open hostility. Lack of affection between the delinquent boys and their parents was noted in every case. The authors concluded that juvenile delinquency results from the complex interworking of a variety of causes.

The items in these letters are contributed by regular correspondents in the various foreign countries.

LONDON

Economy in Drugs.—At a recent Health Services conference in London, details were given of an experiment in the Bristol Royal Hospital that seems to have caused an appreciable saving in the hospital's expenditure on drugs. An economy committee was set up and consisted of a physician, surgeon, pediatrician, pathologist, and pharmacist. The committee met monthly, and at each meeting the cost of particular drugs was discussed. If expenditure on any one item was considered excessive, steps were taken, if possible, to curtail its use. One of the methods used by the committee was to instruct resident medical officers in the correct use of drugs, especially antibiotics, and a booklet was prepared that outlined the essential use of each drug and showed its cost. By careful instruction of nurses, there was a marked saving in the amount of alcohol, iodine, dressings, and antiseptics used in the hospital. It was estimated that the committee saved about £1,000 a year.

Prehistoric Diets.—In the second Albert Reckitt Archaeological Trust lecture before the British Academy recently, Prof. Grahame Clarke made some interesting references to the dietetic and drinking habits of prehistoric man. In north Germany, for instance, in the late glacial period, reindeer was almost the entire source of meat, and in east Yorkshire, during the preboreal phase of the post-glacial period, the red deer was the main source of meat. Evidence obtained from a necropsy on an early Iron Age man from a bog in Jutland showed that he ate a gruel containing barley, linseed, cameline seed, and the fruit of Polygonum lapathifolium. This is similar to the gruel described by Pliny as the common diet of peasants in Greece and Italy. According to evidence from Danish burials, cranberry wine mixed with myrtle and honey seems to have been the drink of the Bronze Age and a brew of barley, cranberry, and bog myrtle in the Roman Iron Age.

Grounds for Sober Optimism.—In the British Medical Association's report on general practice, there was much criticism of the lack of coordination between the different branches of the service. This criticism was justified, but the picture is not one of unrelieved gloom, and in a speech to the Executive Councils' Association the Minister drew attention to examples of cooperation between physicians and the local health authorities. In York, three general practitioners asked for midwives to be allocated to attend prenatal clinics run for their patients in their surgeries. This has been arranged by the local health authority. In Wolverhampton, a coordinated scheme of prenatal care based on a simple procedure has been agreed on between the local health authority and the local general practitioners; this should result in the best use of both the facilities provided by the local authority's prenatal clinics and the services of the practitioners. In Gloucester, a special nursing service has been provided to relieve general practitioners of night visits for certain purposes such as giving subcutaneous injections. Every night, except Sunday, a nurse starts a night round at 8 p. m. to give injections as ordered beforehand in writing by the general practitioners. In Staffordshire, a scheme is in operation under which the general practitioner can refer any chronically sick patient whom he considers cannot be treated at home under prevailing conditions to the Area Medical Officer, who then tries to arrange for home nursing. This has resulted in many patients being treated at home who would otherwise be sent to the hospital.

Influenza Vaccine Trial.—Promising results from the use of an influenza A vaccine during the winter of 1952-1953 are reported by the Medical Research Council committee on clinical trials of influenza vaccine (*Brit. M. J.* **2**:1173, 1953). The trial vaccine was polyvalent with equal proportions of a threefold concentration of F.M. 1 originally recovered in 1947 in the United States and of the egg-adapted Liverpool (1951)

strain. Each dose of 1 ml. contained 9,600 hemagglutinating units of F.M. 1 and 12,000 such units of the Liverpool (1951) strain adsorbed on 10 mg. of aluminum phosphate. The volunteers for the trial were industrial workers, hospital nurses, university students, and the service men. The trial vaccine was given to 6,340 volunteers and the control vaccine to 6,370. Unfortunately, from the point of view of the trial, the incidence of influenza A last winter was comparatively slight, so that the final assessment had to be made on relatively small numbers. These showed, however, that the attack rate of clinical influenza was 3% in those receiving the trial vaccine and 4.9 in those receiving the control vaccine. This gives a difference of 1.9 ± 0.34, a figure that is statistically significant and is equivalent to a 40% reduction in influenza in the trial group compared with the control group. The difference in incidence of febrile cold, febrile sore throat, and other respiratory disease in the two groups was not significant. A comparison of the two groups in different areas of the country according to the incidence of influenza was also made. This showed that in areas of high influenza incidence the attack rate was 3.79% in the trial group and 6.61% in the control group. The incidence of local reactions to the trial vaccine was nil or slight in 97.3%, moderate in 2.6%, and severe in 0.1%. The comparable figures for the control group were 95.9%, 3.9%, and 0.2%. The incidence of general reactions to the trial vaccine was nil or slight in 98.2%, moderate in 1.2%, and severe in 0.5%. The comparable figures for the control group were 97.6%, 1.9%, and 0.5%.

Papilloma Research Scheme.—Details have just been announced of the results of a five year investigation into the incidence and causes of papilloma of the bladder among workers in the chemical industry. This investigation, which has been sponsored by 20 chemical manufacturers, was directed by a scientific committee under the chairmanship of Prof. Alexander Haddow, director of the Chester Beatty Research Institute. The results show that the risk of a tumor of the bladder developing from a "mixed exposure" to alpha-naphthylamine, beta-naphthylamine, and benzidine is about 33 times that of the population at large. Such a risk with beta-naphthylamine is 61 times that of the population at large, the corresponding proportions for alpha-naphthylamine and benzidine being 16 and 19 respectively. The risk appears to be greatest among employees of manufacturers, less among employees of users, and least among refiners. No evidence was obtained to suggest that the manufacture or use of aniline caused bladder tumors. Those employed in the manufacture of magenta and of auramine also showed a significant excess of deaths from bladder tumors. At the Chester Beatty Research Institute, two carcinogenic compounds were isolated as impurities from commercial beta-naphthylamine and a third from a rubber antioxidant derived from naphthylamine. Because of its carcinogenicity, the manufacture of beta-naphthylamine has ceased completely in Great Britain. The replacement of some of the important technical products based on beta-naphthylamine has been achieved by the use of alternative materials as starting points, and, in some instances, by the use of alpha-naphthylamine. It has not been found possible to dispense with the use of benzidine, from which some of the most important dyestuffs are made.

It was recommended that, wherever possible, the carcinogenic material should be delivered to the user-plant as a slurry or solution and that, wherever it is necessary to separate the solid in the dry or paste form for sale to other users, great care be taken to avoid dust in the packing operation. Wooden casks, which could absorb the dangerous material, should not be used or, if they must be used, they should be thoroughly washed immediately after use without steaming or heating.

Wellcome Research Library.—On Dec. 4, Lord Woolton opened the extension to the Library of the Royal Society of Medicine that was made possible by a gift of £125,000 from the Wellcome Trustees. This will allow the library, the largest medical library in Europe, to provide full facilities for the fellows of the society and to house the quarter of a million books that it now possesses. The library was founded in 1806.

PARIS

Experimental Intracardiac Surgery During Artificial Hibernation.—Investigations have recently been resumed to determine the most efficient method of performing intracardiac operations in the open on an exsanguinated heart. The point at issue was the draining of the cardiac cavities without anoxemia of the vital brain centers. Bigelow in 1951 attempted intracardiac operations on dogs with deep hypothermia, because refrigeration reduces the oxygen requirements, but most of these dogs died of shock and others died in the course of the rewarming process. Only 15% survived. On the other hand, Juvenelle, Linde, and Wegelius succeeded in keeping their dogs alive, with the aid of a peripheral heart-lung, at a temperature between 59 and 68 F and with the aorta clamped off for 20 to 30 minutes. This artificial hypothermia is attended by hemoconcentration, acidosis, and hypercoagulability, which are probably an adrenergic response, and later, when endocrine vegetative exhaustion occurs, the action of the cold assumes the form of an irreversible shock. The authors have noted that refrigeration was possible with artificial hibernation achieved by blocking both the central and the peripheral vegetative nervous system and the endocrine system. Instead of hemoconcentration, acidosis, and hypercoagulability, the reverse (hemodilution, alkalosis, and hypocoagulability) was observed. Basal metabolism is much reduced by the blocking drug. The authors thus succeeded in decreasing the temperature of a dog without using ice.

They tried to treat the fibrillations with the method of Prof. Léon Binet and Dr. Strumza, injecting 10 to 20 cc. of a 7.5% solution of potassium chloride into the right ventricle. The solution was continually injected until the heart became inert, inertia being a prerequisite for massage of the heart to restore its rhythmical contractions. Levulose, which is directly utilized by muscular fibers, has often yielded good results. With the method of Binet, the authors obtained 15 good results out of 16 attempts, but the balance achieved was precarious and, above all, the time allowed for work was limited by the duration of the circulatory arrest, which lasted from 5 to 10 minutes, too short a time for a cardiac operation. Eventually they used somatotropin, which in 12 cases stopped all fibrillations during clamping for 15 to 20 minutes. This enabled them to operate on an animal with a temperature of 82.4 F. To prevent late fibrillation that occurs a few minutes after declamping, they declamped the vena cavas separately and gradually at intervals of a few minutes. A high degree of superoxygenation is indispensable at that stage.

After some groping the following technique has been devised: The day preceding the operation, the animal is given an injection of 20 to 40 units of somatotropin. On the morning of operation a similar injection is given. Hibernation is then brought about by means of a "lytic cocktail," consisting of 0.75 gm. of Largactil (dimethylaminopropyl-N-chlorophenothiazine hydrochloride), 0.05 gm. of Phenergan (10-[2-dimethylaminoisopropyl] phenothiazine), and 0.05 gm. of meperidine (Dolosal) in 6 cc. of isotonic sodium chloride solution and administered intravenously every 15 minutes for at least 90 minutes. While hibernation is being established, the animal is given an intravenous injection of 5 to 10 units of somatotropin. Under the influence of the latter the animal, which is still agitated, falls asleep. The drop in temperature is gradual and the animal no longer presents any defense reaction. No jerks of the extremities or clonic spasms are seen. The rhythm of the heart, accelerated by "the cocktail," slows down under the action of somatotropin.

When the thorax has been opened, the authors inject procaine into the pericardial cavity. The recorded cardiac rhythm is characterized by a slowing and by a substantial increase in pulse pressure. It is not necessary to give the animals heparin because the coagulability of their blood is decreased as result of adrenergic blocking. Warming is started as soon as the thorax has been closed. Drug perfusions are discontinued, the animal is placed on a litter with its head slightly downwards,

and antibiotics are given. The closing of the thorax requires special care. The lungs must not be injured because the dog has only one pleural cavity. The azygos vein must be ligated to drain the cardiac cavity.

Comparative Studies of Vascularization in Tuberculous Cavities and in Limited Caseous Foci.—On May 9, 1953, J. Delarue, C. Sors, and J. Mignot addressed the French Society of Tuberculosis on the results of their investigations carried out with the aid of arterial, bronchial, and pulmonary injections and radiography. They found an obstruction of pulmonary arteries in tuberculous cavities. At this level vascularization is taken over by the bronchial vascular system. As regards tuberculomas, anastomoses seem to be less numerous, and at some points the vascular patterns are similar to those found in the cavities. At such points the caseous material seems to be undergoing liquefaction. The authors showed that the liquefaction of caseous material is closely linked with a change in local circulation.

SWEDEN

Modern Treatment of Tuberculosis.—The combination of chemicals with antibiotics in the treatment of tuberculosis is showing much the same pattern in Sweden as in various other countries, but, with so many drugs and combinations of them and with such a wide range of dosage and time-spacing, this pattern is apt to change in detail from time to time. The inclusion of penicillin in the pattern is one of the special features of the policy adopted by the medical department of the Serafimer Hospital in Stockholm, under the charge of Prof. Anders Kristenson. His advocacy of penicillin in this connection is prompted by his suspicion that secondary infections may play an important part in pulmonary tuberculosis, particularly when it has reached the stage of cavitation. In vitro tests by Espersen have suggested that the combination of penicillin with streptomycin is beneficial by virtue of a synergistic action on the tubercle bacillus. The following scheme lasting four weeks is recommended for adults by Professor Kristenson: Every day 1 gm. of dihydrostreptomycin, 300,000 international units of penicillin, 0.3 gm. of isoniazid in three doses, and 12 gm. of *p*-aminosalicylic acid (PAS) in three or four doses. At the end of this period, the isoniazid and *p*-aminosalicylic acid are given daily as before and the same dose of streptomycin and penicillin is given only twice a week. Changes are introduced in this procedure in response to the behavior of tubercle bacilli in the sputum, including their resistance to the drug used. Changes in the radiological findings are also a guide to treatment, and the dosage of *p*-aminosalicylic acid has sometimes to be changed when it provokes complications. Professor Kristenson foresees a time when it may be possible to give a combination of the above-mentioned drugs early in the disease with the hope of sooner being able to let nature heal the lesions.

Dr. Gösta Birath of Gothenburg believes that damage to certain cranial nerves due to dihydrostreptomycin may be less severe in Sweden than elsewhere because of the avoidance of prolonged and uninterrupted treatment with this antibiotic. He is in favor of greater spacing between each injection of dihydrostreptomycin, which is now injected, as a rule, only two or three times a week. In choosing between streptomycin and dihydrostreptomycin for infections other than tuberculosis he prefers the latter because it is less likely to provoke allergic phenomena in patients and hospital staffs, whereas the risk of damage to the auditory nerve is about the same for both drugs when they are given over a comparatively short period of time.

Legal Abortion Permitted but not Practiced.—Abortions, accidental, legal, and criminal, continue to attract much attention in the Swedish press, both medical and lay. In *Svenska Läkartidningen* for Nov. 20, 1953, Dr. Per Arén of Uppsala and Dr. Curt Åmark of Stockholm have asked how the mothers who have been granted the legal induction of abortion, but, have changed their minds and have let their pregnancies go to term fare. The 32 women who followed this course were interviewed in their homes an average of three and a half years after the confinement in question. The gynecologic and psychiatric examinations they underwent indicated that the course they had taken was in most cases justified. Three of the infants had died at birth or shortly thereafter, and three others had been adopted by foster parents as soon as they were born. The remaining 26 mothers had cared for their own babies, and at least 20 were leading well-adjusted and harmonious lives. Only three women had regretted the lost opportunity to accept a legal abortion. This follow-up study suggests that legal abortion is apt to be permitted unwisely and unadvisedly even when the safeguards provided by the law have seemingly been effective. During the follow-up examination, the mothers were questioned about the indications for the induction of legal abortion and in 11 cases information on this subject was not obtained. In most of these cases conflict situations may have existed. In 25 cases social and economic difficulties had been invoked as a plea for the induction of abortion, and at least 12 women had again become pregnant. It was a curious and instructive psychological phenomenon that some of the women experienced a sense of relief on learning that the authorities had consented to abortion. Whatever the explanation, this change of mind points to the wisdom of allowing time and opportunity for the woman to reconsider her own case. In some cases abortion had been permitted on eugenic grounds on the strength of intelligence tests, the result of which may be misleading under the strains of pregnancy. In several cases the sense of depression and debility experienced by the applicants for abortion had proved to be transient; another reason why this indication for abortion should be considered most critically.

Prognosis for Sarcoidosis.—A curious outcome of mass radiographic examinations for tuberculosis has been an impressive series of cases of sarcoidosis, many of which would not have come to light but for this test. The information thus obtained may help to solve the riddle of the pathogenesis of this disease and give clues to the prognosis for it. From the medical department of the Falu Hospital comes a report by Dr. S. E. Fagerberg whose study covers the period 1944 to 1951 and deals with 58 patients observed among a total of 18,432 admitted to this hospital. After the omission of 4 patients there remained 54 (25 men and 29 women) about whose subsequent fate information was available. The ages of these patients ranged from 20 to 69 years. In addition to the 7 patients who had died, 10 were found on reexamination to be subjectively and objectively well and 9 felt well but still showed objective signs of the disease, such as changes in the lungs, eyes, or bones. The remaining patients still presented various subjective and objective manifestations of the disease. In 5 patients involvement of the lungs or eyes entailed severe invalidism, and in 32 there was impairment of health but not severe invalidism. In view of the comparatively short observation period and the prospect of progress of the disease in some of these patients, Dr. Fagerberg is dubious over the prognosis of a disease that has been given the somewhat misleading name of benign lymphogranulomatosis. With only 10 patients showing both subjective and objective recovery on reexamination, the adjective benign seems to be a misnomer.

Another report on sarcoidosis comes from Dr. Hanns G. Bauer whose material consists of 185 patients with pulmonary sarcoidosis discovered in the course of about 423,000 mass radiographic examinations in Stockholm. His report, published in *Nordisk Medicin* for Dec. 3, 1953, shows that most of these patients were between the ages of 25 and 40 years, none were under 18, and the disease was rare in old age. The experience of the past 10 years suggests that the radiological manifestations of pulmonary sarcoidosis can be classed in three main groups showing marked differences with regard to the prognosis. The prognosis seems to be worst when the radiological manifestations indicate involvement of the lung parenchyma. Most of these 185 patients were referred from the Stockholm Central Tuberculosis Dispensary to the St. Göran Hospital, where it was found that the patients were often impatient of control as the absence of subjective symptoms made it difficult for them to realize that they were ill. But for mass radiography, many of them would have remained unconscious of their sarcoidosis.

CORRESPONDENCE

VISUAL AIDS FOR TEACHING

To the Editor:—At the first World Conference on Medical Education in London, the section on teaching methods devoted considerable time to papers and discussions of visual aids. It was stressed that, although a great deal of money is spent on photography, films, and other visual aids, there is little critical evaluation of the aids in the literature or at medical meetings.

For the past several years I have used colored slides to amplify the teaching of the acute exanthems and would like to record my experience with this method in order to get some exchange of opinion or discussion into the literature.

To test the accuracy of reproduction of the first 100 slides purchased I reviewed them and discarded 60 because the condition was not recognizable without a look at the label. It should be obvious that, if an experienced teacher does not recognize the picture as reproduced, the inexperienced student can get little of permanent value from the slide. In teaching from slides instead of from the patient, certain limitations of the method must be borne in mind by the teacher. In all photography the picture conveys much more to the person who has seen the original than to one who has not. It was surprising, for instance, to have a student ask whether the rash was elevated or flat when a slide of erythema nodosum was flashed on the screen. If the rash has not been seen, it is very difficult for the student to interpret correctly factors such as shades of color. It is also difficult to determine whether a rash is fine, feels granular, blanches on pressure, or has any depth to it. If the picture is taken of the entire body, then the details are missed; if a close-up is taken of only a portion of the rash, the picture as a whole is lost. In some diseases, it is better to supplement a still picture with a short movie that not only shows the distribution over the whole body in a long shot but also portrays the more intimate details of the rash by several close-ups. It should be noted that such manifestations as Koplik's spots or an acute sore throat are much better portrayed by a short movie than by a single still picture. When the first slides were collected it was thought that one good slide of a classic picture was all that was necessary. Experience has shown, however, that many slides showing variations are far more effective teaching material.

In the order of their usefulness, slides of chickenpox, smallpox, and vaccinia lend themselves best to this method of teaching. Most of the students have seen chickenpox, a vaccination, or at least a vesicular hive, so there is some personal experience on which to elaborate the discussion. Furthermore, the pox photographs well. It is discrete, and often the elevation is shown when caught in the lateral view. With several slides on hand it is possible first to flash one on the screen and ask for a diagnosis. This may be followed with a question like "What day of the disease is represented?" Then several slides may be shown indicating variation, distribution, and severity and leading up to a comparison with a series of smallpox. Similarly several slides of a vaccination can show the development of the lesion from day to day. The immune reaction or vaccinoid can be compared with the primary take. Slides bring out questions such as differentiation of a severe take from one secondarily infected. The question may be raised "Do satellites produce scarring?" Pictures of generalized vaccinia of different types can be shown, and, finally, the picture of a case of eczema vaccinatum gives an opportunity to discuss the contagious nature of vaccinia to unvaccinated eczematous infants. Used this way the slides have been practical and helpful.

The next disease that can be fairly well demonstrated by color slides is meningococcemia. The ecchymosis and petechiae photograph readily. The lesion is in the skin and does not call for evaluation of texture or depth. Several slides portraying variations from the mild to the severe with extensive ecchymosis give the student a picture of this disease that is not easily forgotten, even if he has not had the opportunity to see a patient. Since meningococcemia is one of the few medical emergencies, it is very worth while to discuss it with the help of slides. Other petechial diseases or purpuras may be shown for comparison.

For the most part measles is less satisfactorily portrayed by slides than the other diseases. A well-taken picture may convey much to a teacher, but a student who has never seen measles may miss most of the significant findings. The rash of measles has a color and texture that are impossible to wholly catch by photography. Unfortunately the two diseases from which measles must be differentiated, rubella and exanthem subitum, photograph very poorly and make it almost impossible to contrast the three diseases by pictures. In spite of these handicaps, slides on measles can be used to some advantage, as to portray distribution, confluence, and the morbilliform nature and to contrast it with scarlet fever even though the latter does not photograph convincingly. The rash of scarlet fever has a texture and color that is not readily portrayed. It is difficult to demonstrate the blanching, and the marked variation from one area of the body to another is not readily shown by slides. On the other hand, the tongue of a patient with scarlet fever photographs very well. It is also worth while to have a good picture of desquamation since it is infrequently seen now.

It should be added that research in the use of visual aids should not be left to camera fans or reluctant teachers who need props. It should be diligently analyzed by objective critical observers and educators.

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LEPROSY

To the Editor:—Dr. William Feldman's suggestion (*J. A. M. A.* **153:**1041 [Nov. 14] 1953) that Mycobacterium leprae be renamed M. hansenii, so that the disease, leprosy, may be renamed "hansenosis," has certain disadvantages to which attention should be drawn. 1. Mycobacterium leprae and leprosy or *lepra* or *la lèpre* are worldwide expressions, whereas the objectionableness of the word leper and the debatable objectionableness of the word leprosy are, as Roland Chaussinand of the Institut Pasteur in Paris has pointed out, purely Anglo-Saxon problems. 2. Leprosy, whatever its etymology, has been applied to this disease in English speaking areas for over six centuries; this is a stronger argument for its correctness than the names of many diseases can offer. 3. The word leper is admittedly objectionable, and its avoidance was urged by both the Fifth and the Sixth International Congress of Leprosy; yet both congresses recommended retention of leprosy as the name of the disease. 4. General, unemotional public acceptance of syphilis, cancer, and tuberculosis has been achieved for these three once horrifying diseases by talking and writing about them under their proper scientific names. Could this have been done as well, or at all, if they had been renamed "schaudinnosis," "virchowosis," and "kochosis"? I think it highly unlikely: their old names would have continued to inspire the same old emotional reactions once they became known. So will the name leprosy, once it is used to explain what "hansenosis" means. Dr. Feldman's suggested short cut may bypass a tedious program of public education, but it will prove in the long run to follow a circular path that winds up about where it started.

My sympathy and concern for the feelings of the victims of leprosy are genuine. I feel it would be a serious disservice to them to outlaw the name of their disease—as if it were so dreadful an affliction that its name could not even be uttered aloud. Rather let us use it freely, openly, unemotionally, objectively, and properly, until its sound evokes, like the sound of the word tuberculosis, only the picture of another potentially communicable disease that can be controlled by proper medical treatment.

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COUNCIL ON MEDICAL SERVICE

The Committee on Medical Care for Industrial Workers, a joint committee of the Councils on Medical Service and Industrial Health of the American Medical Association, has been conducting a study of health centers established and operated by labor unions. The data on 12 such centers are contained in a recently published report, available to those interested in the subject. The following is a brief summary of the material contained in this study.

UNION HEALTH CENTERS

Union health centers are a comparatively recent development in the United States. Several industries have had medical service plans for their employees for almost a century, but the earliest union center on record is that of the International Ladies Garment Workers Union in New York City, organized in 1913. This plan, however, was the only union venture in the health field for some 25 years. Current union interest in health benefits arose in 1942, when the federal government's wartime anti-inflation measures practically halted wage increases. The War Labor Board ruled that a welfare plan agreed upon by employer and union would not be considered a wage increase if the cost did not exceed 5% of the payroll. Since employers were allowed to deduct their share of the cost of such plans as a legitimate business expense, health and welfare plans became of prime importance in labor-management negotiations. When wage controls were discontinued, the interest in health plans remained. At latest count, these "fringe benefits" have become part of the employment contract of some 16 million workers, most of whom are covered through recognized insurance procedures. The main exceptions to this rule are the program of the United Mine Workers of America, which pays physicians and hospitals directly for services rendered UMWA members and their families, and the various "union health centers," which provide primarily diagnostic services to union members and their families.

The Committee report describes 12 of these centers, with details concerning organization and operation. They vary in size and location and include both C.I.O. and A.F.L. affiliate sponsored plans. The data have been classified under the following headings: (1) history, (2) membership, (3) objectives, (4) facilities, (5) financing, (6) budget and operating costs, (7) administration, (8) business and professional personnel, (9) benefits, and (10) preventive aspects of the services. So far as possible, the same headings are used in this summary. The Committee has not evaluated any of the plans. However, the present factual study lays a foundation for such evaluation in the future.

TABLE 1.—*Centers Studied*

Plan	Begun	Location	Union
AFL Medical Service Plan	1951	Philadelphia	AFL
ILGWU Union Health Centers	1913	New York	AFL
	1944	Philadelphia	
	1952	Cleveland	
	1950	Minneapolis	
	1950	Kansas City, Mo.	
	1947	St. Louis	
Labor Health Institute	1945	St. Louis	AFL
Hotel Trades Health Center *	1950	New York	AFL
Sidney Hillman Health Centers	1951	New York	CIO
	1951	Philadelphia	
UAW (United Auto Workers) Health Institute	1942	Detroit	CIO

* Full title: Hotel Trades Council and Hotel Association Health Center, Inc. Abbreviated title will be used in all tables.

The ILGWU has a number of centers throughout the country not included in this survey. It is believed, however, that the six studied thus far are representative of the types operated by this union.

HISTORY

The Union Health Center of the International Ladies Garment Workers Union, founded in New York in 1913, was not only the earliest of these centers but also, for many years, the only one. The next development was the UAW Health Institute, begun in 1942, almost 30 years later. By 1950, three more were in operation. Half of the total number studied were not begun until 1950, 1951, and 1952. The stimulus of the government wage "freeze" is obvious, since only two began before controls went into effect. The first ILGWU Union Health Center began in a different way from its successors. Founded before the first World War, at a time when wage increases were more important than "fringe" or "security" benefits, it was financed entirely by members' contributions. The first center consisted of two small rooms, served by one physician with limited equipment, and was designed primarily as a measure against tuberculosis, then almost an occupational disease in the garment industry. Through the years, the center grew in size and in services provided; collective bargaining brought employer contributions to help in financing. When wage controls began, the advantages of this health center to union members had been thoroughly demonstrated; it became, in a sense, a model not only for ILGWU locals in other cities but also for other unions seeking benefits to replace prohibited wage increases.

MEMBERSHIP

With the exception of the Labor Health Institute, center membership automatically includes all those eligible for care. Union membership means health center membership. The Labor Health Institute estimates that a little less than half those eligible are enrolled, since the number eligible includes dependents who are not yet covered by collective bargaining agreements. The number of union locals and members eligible for each center is shown in the following table:

TABLE 2.—*Eligible Membership*

Plan	Locals	Members
AFL Medical Service Plan	14	14,000
ILGWU Union Health Centers		
New York	32	202,000
Philadelphia	5	19,000
Cleveland	17	5,000
Minneapolis	5	800
Kansas City	8	3,000
St. Louis	13	4,500
Labor Health Institute	1	15,000
Hotel Trades Health Center	10	33,668
Sidney Hillman Health Centers		
New York	26	33,356
Philadelphia	16	23,000
UAW Health Institute	170	350,000
Totals	317	703,324

The number of members actually treated at a center during a year generally ranges between a third and a quarter of those who are eligible. The main exceptions are the ILGWU center in Minneapolis, which in 1952 served about 350 of its 800 members, and the UAW Health Institute, which in 1951 served only 16,000 of the 350,000 eligible.

OBJECTIVES

Most centers were developed primarily to provide a complete diagnostic ambulatory clinic. They have also emphasized, with not too great success, preventive medicine to promote workers' health and to avoid loss of income due to illness. One plan (the Hotel Trades Council and Hotel Association Health Center, Inc.) includes hospital service; in that center physicians will treat hospitalized members at reduced rates. The Labor Health Institute provides hospital care by staff physicians and, in addition, some home care. Few centers have definite plans for adding to their services, although several of the administrators believe that, in the future, some provision will have to be made for coverage of such uninsured medical expenses as home care, drugs, and nursing services. At present, however, most centers are concentrating on original objectives, working to increase efficiency and utilization by the members.

FACILITIES

Some idea of the facilities available at the various centers may be obtained from the following tabulation of facilities and costs:

TABLE 3.—*Health Center Facilities*

Plan	Facilities
AFL Medical Service Plan	Section of 1st floor in hospital
ILGWU Union Health Centers	
New York	6 floors, 100,000 sq. ft.
Philadelphia	2 floors, 14,000 sq. ft.
Cleveland	2nd floor of mansion
Minneapolis	2nd floor office, 7 rooms
Kansas City	3 story house
St. Louis	3 story house
Labor Health Institute	3 floors in office building
Hotel Trades Health Center	5 story office building
Sidney Hillman Health Centers	
New York	6 story building, 36,000 sq. ft.
Philadelphia	2 story building, 45,000 sq. ft.
UAW Health Institute	2 floors of 3 story building

TABLE 4.—*Health Center Costs*

Plan	Cost (in Thousands)			
	Construction/ Purchase	Rent (Ann.)	Remodeling	Equipment
AFL Medical Service Plan	$.....	$4.0	$ 62.0	$ 66.0
ILGWU Union Health Centers				
New York	2,500.0*	...	892.0	.....
Philadelphia	250.0	...	.....	85.0
Cleveland	.....	4.8	82.5	51.0
Minneapolis	.....	1.8	10.0	13.0
Kansas City	35.0	...	32.0	32.6
St. Louis	35.0	...	34.8	35.8
Labor Health Institute	135.1	...	138.0	85.8
Hotel Trades Health Center	96.5†	...	551.8†	126.6†
Sidney Hillman Health Centers				
New York	110.0	...	500.0	120.0
Philadelphia	1,086.0	...	.....	183.6
UAW Health Institute	50.0	...	82.0	46.5

* Cost to union of entire 27-story building; center uses 6 floors.
† Cost analysis, Hotel Trades Health Center: "Land, $96,470; building and fixed equipment, $551,767; movable equipment, $126,630."

Neither the size of the center nor the amount of money spent will give an accurate indication of its worth. They do indicate, however, that an earnest attempt is being made to provide the modern and complete facilities necessary for good clinical services. In general, the centers are well equipped and housed in space adequate to the size of the groups served.

FINANCING

As previously mentioned, the New York ILGWU center was at first financed entirely by union funds; however, later collective bargaining agreements brought employer contributions. At present, the UAW Health Institute is the only plan studied that received no financial aid from management. The remaining plans were begun through collective bargaining agreements and, in most of them, the employer pays the entire cost. The payment, varying from 1% to 5% of the gross payroll, is usually made to the union local, which then pays the center a flat rate per eligible member. In some cases, the Sidney Hillman Health Centers for example, employer and employee share the operating costs. In another case, union funds built the center, and employers finance its operation. The UAW Health Institute is financed by flat rate per member levied on each participating local union. The rate decreases for locals more distant from the institute. Since many of the health funds began during the "wage freeze," some unions feel the employer contribution is in lieu of a wage increase and that, indirectly, the center is entirely union-financed.

BUDGET AND OPERATING COSTS

The UAW Health Institute has no year-to-year limitation on expenditures; the international union Health and Welfare Fund provides the amount necessary to operate the institute. The other centers operate on a budget basis. Most plans either have a reserve fund or are planning one as soon as initial indebtedness is met. Total operating expenses and, where available, the amount used to meet salaries or fees of physicians and auxiliary personnel are tabulated below:

TABLE 5.—*Operating Expenses*

Plan	Operating Expenses	Fiscal Year	Physicians' Payments
AFL Medical Service Plan	$ 81,700	1951	$ 39,000
ILGWU Union Health Centers			
New York	1,484,051	1952	491,954
Philadelphia	168,000	1951	119,000*
Cleveland	36,392	1952	.....
Minneapolis	14,000	1952	.....
Kansas City	62,706	1952	.....
St. Louis	111,422	1952	.....
Labor Health Institute	865,142	1952	.....
Hotel Trades Health Center	586,549	1952	260,600
Sidney Hillman Health Centers			
New York	327,000	1952	116,000
Philadelphia	375,502	1952	172,731†
UAW Health Institute	367,174	1951	.....

* Physicians and auxiliary personnel.
† Listed as "about 46% of operating expenses."

ADMINISTRATION

Of the 12 centers, 4 have only union representatives on the board of directors. The AFL Medical Service Plan has two board members from each local and three officers from the central labor union; the UAW Health Institute board of trustees is composed of five members of the UAW's executive committee, including the president. The New York ILGWU center's board is also composed of union officials, while the Philadelphia center has a director from each of the five affiliated unions. The eight remaining centers have both union and management directors. The Sidney Hillman Health Center in New York has a 21-man board, elected jointly by unions and managements. The center in Philadelphia has 12 representatives each from unions and employers. The Hotel Trades center is directed by 10 representatives each from the Hotel Trades Council and the Hotel Association. The Labor Health Institute is unique among the 12 plans in that, in addition to 3 management and 20 union board members, it also has 2 public representatives. The 25-member board elects a 6-member executive committee to operate the institute. Each center has a full or part-time medical director for medical administration. Larger centers have medical advisory boards, composed of the chiefs of services or of staff members. The board of directors is generally responsible for over-all policy divisions and usually for appointment of the medical staff, although the choice may be made from a list submitted by the medical advisory board.

BUSINESS AND PROFESSIONAL PERSONNEL

Administrative personnel vary greatly in number, from 3 in one center to 144 in another; in most centers, 1 office worker is employed for approximately 1,200 members. Some of these employees work only part time, and, in some centers, they act as clinical assistants or maintain health records not directly related to functions of the center itself. Most of the physicians serve the centers on a part-time basis. Some hold sessions at irregular intervals, some are at the center two to four hours daily, some are available on a consultation basis. Aside from medical directors, the only fair-sized staff of full-time physicians is at the New York ILGWU center, which has 10. Few of the other centers have more than one. Most centers employ only specialists. The four with general practitioners are the New York ILGWU center, the Hotel Trades center, the New York Sidney Hillman center and the UAW Health Institute. Payment to physicians is usually a flat rate per hour, ranging

from $4.00 to $12.00. Several centers have a graduated increase in fees, depending on the length of service at the center. One has a different rate for general practitioners and specialists, and one has a highly complicated schedule that bases the specialist's rate per hour on the median annual income of specialists in his field. Two centers pay on a fee-for-service basis, while at the New York ILGWU center, full and part-time physicians are on an annual salary and the consultants on a per-hour basis. The following table shows the total personnel of each center, full and part time (including consultants), and the method of physician payment.

TABLE 6.—*Personnel*

Plan	Personnel				Physician Payment
	Administrative	Physicians	Nurses	Auxiliary	
AFL Medical Service Plan...	10	54	5	3	$7 per hr.
ILGWU Union Health Centers					
New York.....	144	175	15	74	Sal. & per hr.
Philadelphia.....	15	23	4	4	$7 per hr.
Cleveland.....	3	34	1	11	$10 per hr.
Minneapolis.....	4	1	0	1	Fee basis
Kansas City.....	3	21	1	2	$10 per hr.
St. Louis.....	6	17	1	2	$6-12 per hr.
Labor Health Institute.....	20	58	16	12	Specialty rate
Hotel Trades Health Center	49	183	12	12	Fee basis
Sidney Hillman Health Centers					
New York.....	23	81	4	8	$5-7½ per hr.
Philadelphia.....	28	44	5	12	$7 per hr.
UAW Health Institute.....	16	34	1	9	$4-6 per hr.

BENEFITS AND PREVENTIVE ASPECTS OF SERVICE

In general, all centers provide the same type of service; their primary objective is complete ambulatory diagnostic care. Completeness, of course, varies with the size of the center and the amount of its funds. A few centers, the larger ones particularly, provide therapeutic care, with some emphasis on physical therapy. Several centers operate their own pharmacies and provide prescriptions at cost or at a reduced rate. As previously mentioned, two centers have arrangements for hospital care by staff physicians, and one provides some home care. Physicians at the New York Sidney Hillman Center have agreed to accept the fee schedule of the union's commercial surgical insurance in full payment for hospital care of members, thus providing an informal service benefit plan. Several centers provide special services beyond ordinary diagnostic care. The UAW Health Institute, for example, provides optometric service and a reduced rate on glasses for members. It also operates a mental hygiene service for the entire community with partial support from the community chest. All centers cooperate with members' private physicians, sending them reports on tests or examinations conducted at the center. Several centers have invited local physicians to use their diagnostic facilities. All centers are interested in preventive care, the method varying with the resources of the center. Some sponsor health education talks in plants or at the center; some distribute posters and bulletins on health topics. All emphasize the importance of regular check-ups, and one center is experimenting with multiple screening, with a mobile unit planned for visiting the various plants.

COMMENT

Despite minor variations, the union health centers studied represent definite union experimentation in the medical care field. As yet, most medical coverage is on an insurance basis, but unions are using "the high cost of medical care" and the exclusion of diagnostic services from most insurance policies as an argument for union-sponsored health centers. Thus far, the centers have concentrated on diagnosis, but expansion may well include treatment in the home and possibly in the hospital as well as at the center. In general, the centers seem modern and well equipped. A major problem for most of them is insufficient utilization by members, partly because of difficulty in visiting the center during working hours. To provide increased utilization some plans permit, or are planning to permit, the members' dependents to use the facilities. No evaluation of plans or comparison between centers has been made. However, the continued union interest in such plans, even after the removal of wartime wage controls, indicates the need for further study to determine standards by which they may be evaluated.

BUSINESS PRACTICE

The following material is based on a public relations manual issued by the Public Relations Department of the American Medical Association.—ED.

GETTING CREDIT INFORMATION

Collection begins with the first contact with the patient. Each experience a patient has in the doctor's office lays the groundwork for future payment. Because good records are essential to follow-up collections, it is important to get full information from patients on their first visit if possible. It is neither unprofessional nor greatly time consuming to obtain credit information from patients. In about 60 seconds a physician or his secretary can ask a few questions that make possible a shrewd appraisal of a patient's ability to pay. The information that should be obtained includes:

1. The patient's full name, correctly spelled. J. A. Brown is not sufficient; John Allen is more helpful. If full names are not taken, it may be impossible to locate the J. A. Brown treated in later collection follow-ups.

2. The name of the person to be billed as well as the relationship to the patient, i. e., wife treated, husband billed.

3. Marital status and number of dependents. When couples are divorced or separated, credit and collections become complicated. Inquire of a woman, for example, whether the bill should be sent to her husband at such and such an address.

4. Current address and past addresses for at least the past two years. From this information the person's stability can be determined, i. e., many residences in a short period of time may indicate financial instability. The type of neighborhood may also indicate a patient's financial status.

5. Occupation of responsible person. If a man says he works at Gordon's department store, find out whether he is a shipping clerk, a salesman, or a vice-president.

6. Who referred the patient. It is wise when referring a patient to another doctor to send along credit information or data about the patient's ability to pay.

7. The patient's charge accounts. These give good credit information. Do not ask about banks and bank accounts, as this sounds too mercenary or gives too great an impression of prying into personal affairs.

8. If a large fee is involved, a tactful inquiry about the patient's approximate annual income and financial responsibilities may be made in order to set an equitable fee.

References should not be asked for. Persons rarely list references who will be inclined to give a truthful answer anyway. Superficial signs of prosperity are often misleading. A patient who appears to be very wealthy may owe bills all over town. If a patient's ability to pay is questionable and a large fee is involved, a local credit agency can be consulted. There are over 1,200 associated credit bureaus in America offering a great deal of credit information. If the local medical society maintains a bureau of medical economics, it often can help determine a patient's credit rating. It is easier for a doctor in a small town to gage informally his patient's ability to pay. In a large city the task is more difficult and must be pursued in a more formal way, but an approximate determination of a person's ability to pay is necessary in order to provide medical care for all.

MEDICAL LITERATURE ABSTRACTS

UNITED STATES

A.M.A. Arch. Ind. Hyg. & Occupat. Med., Chicago

8:391-492 (Nov.) 1953

Fear, Frustration, and Futility: Industrial Aspect. R. T. Johnstone. —p. 391.
Toxicity of Malathon: Summary of Mammalian Investigations. L. W. Hazleton and E. G. Holland.—p. 399.
Studies in Vanadium Toxicology: Reduction in Cystine Content of Rat Hair. J. T. Mountain, L. L. Delker and H. E. Stokinger.—p. 406.
Absolute Method of Sampling and Measurement of Solid Air-Borne Particulates: Combined Use of Molecular Filter Membrane and Electron Microscopy. D. A. Fraser.—p. 412.
Silicosis in Mica Mining in Bihar, India. H. Helmann, S. Moskowitz, C. R. H. Iyer and others.—p. 420.
Some Physiological Effects of Disulfur Decafluoride (S_2F_{10}) After Intravenous Injection in Dogs: Note on Its Toxicity to Rabbits. J. P. Saunders, M. Shoshkes, M. R. De Carlo and E. C. Brown.—p. 436.
Dust Sampler which Simulates Upper and Lower Lung Deposition. W. B. Harris and M. Eisenbud.—p. 446.
Pneumoconiosis in Tungsten-Carbide Tool Industry: Report of Three Cases. C. W. Miller, M. W. Davis, A. Goldman and J. P. Wyatt. —p. 453.
Studies of Acute and Chronic Toxicity of Germanium. G. Rosenfeld and E. J. Wallace.—p. 466.
Absence of Toxic Manifestations in Workers Exposed to Chlordane. W. C. Alvarez and S. Hyman.—p. 480.

A.M.A. Arch. Neurology and Psychiatry, Chicago

70:551-686 (Nov.) 1953

Studies on Headache: Long-Term Observations of Reactivity of Cranial Arteries in Subjects with Vascular Headache of Migraine Type. M. M. Tunis and H. G. Wolff.—p. 551.
Factors that Influence Prognosis in Acute Focal Cerebrovascular Lesions. C. H. Millikan and F. P. Moersch.—p. 558.
*Failure of Abnormal Cerebrospinal Fluid Pressure to Influence Cerebral Function. H. W. Ryder, A. Rosenauer, E. J. Penka, F .F. Espey and J. P. Evans.—p. 563.
Some Relationships of Intelligence, Mental Efficiency, Mental Deterioration, and Disease. M. P. Manson.—p. 587.
Pituitary-Adrenal Cortex Reactivity in Schizophrenic Patients. W. F. Fry Jr.—p. 598.
Central Inhibition in Cortical Conditioned Reflexes. F. Morrell and M. H. Ross.—p. 611.
Statistical Control Studies in Neurology: IV. Discomfort with Conjugate Gaze in Head Injuries. N. Savitsky and M. J. Madonick. —p. 617.
Ciliospinal Reflex in Injuries of Cervical Spinal Cord in Man. A. J. Arieff and S. W. Pyzik.—p. 621.
Hallucinations in Braille: Effects of Amygdaloidectomy. W. Freeman and J. M. Williams.—p. 630.
Effect of Carbon Tetrachloride on Nervous System. H. Stevens and F. M. Forster.—p. 635.
Factors Influencing Extensor Tonus and Respiration in Decerebrate Cats. W. D. Hagamen and R. L. Beals.—p. 650.

Cerebrospinal Fluid Pressure.—Several hundred patients with and without abnormal cerebrospinal fluid pressures and disorders of the central nervous system were submitted to lumbar puncture with addition to and subtraction from the fluid in order to study the relation between the fluid pressure and changes in craniospinal blood volume. Of these patients, 157 also had lumbar barographies in which fluid was withdrawn from the lumbar subarachnoid space, reinjected, and then enough withdrawn to return the pressure to the original level; at the same time, the internal jugular venous pressure, the internal carotid or brachial artery pressure, and usually the respirations were recorded. Diverse reactions to this procedure included various types of headache on both addition and subtraction of fluid, painful restlessness on addition of fluid, and a "Cushing reaction" (rise in blood pressure and in jugular pressure and acceleration and deepening of respiration) in the few patients in whom the pressure was raised to the level of the mean arterial blood pressure. Induction of these reactions associated with extreme deviations in fluid pressure could in no case be shown to be directly due to the induced change in pressure. The mechanisms of the reactions are discussed. It is noteworthy that lumbar barography does not produce the nausea and vomiting associated with ventriculography and encephalography. The authors conclude that, since the cerebrospinal fluid pressure has no direct effect on cerebral function, there are no absolute pressure receptors in the brain. The factor that does modify cerebral function is believed to be the stretching or distortion of craniospinal structures that stimulate afferent nerve fibers and thus produce the symptoms associated with changes in fluid pressure.

Alabama State Medical Assn. Journal, Montgomery

23:91-118 (Oct.) 1953

Toxemia of Pregnancy. J. C. Carmichael.—p. 91.
Rectal Carcinoids. E. G. Bovill and E. H. Roberts.—p. 93.
Diagnosis and Management of Irritable Colon. A. W. Alford.—p. 99.
Some Indications for Sympathetic Block. F. Nicholson.—p. 105.

American Journal of Obstetrics & Gynecology, St. Louis

66:929-1160 (Nov.) 1953

Optional Nursery Facilities for Care of Mothers and Newborn Infants. J. Parks, P. A. McLendon and M. Kelley.—p. 938.
What are the Earliest Endometrial Changes to Justify Diagnosis of Endometrial Cancer? R. W. Te Linde, H. W. Jones and G. A. Galvin. —p. 953.
*Pregnancy Associated with Disease of Adrenal Glands. A. B. Hunt and W. M. McConahey.—p. 970.
Vascular Bed of Bulbar Conjunctiva in Normal Menstrual Cycle. R. Landesman, R. G. Douglas, G. Dreishpoon and E. Holze.—p. 988.
Relationship of Hormonal Environment to Genesis and Inhibition of Neoplastic Growth: Is Cancer Autonomous? R. W. Rawson.—p. 999.
*Cerebral Circulation and Metabolism in Toxemia of Pregnancy: Observations on Effects of Veratrum Viride and Apresoline (1-Hydrazinophthalazine). M. L. McCall.—p. 1015.
Is Tracheotomy Indicated in Eclampsia? C. G. Collins, F. G. Nix and I. Dyer.—p. 1031.
Mechanisms of Bleeding During Pregnancy. R. A. Bartholomew, E. D. Colvin, W. H. Grimes, J. S. Fish and W. M. Lester.—p. 1042.
Does Administration of Diethylstilbestrol During Pregnancy Have Therapeutic Value? W. J. Dieckmann, M. E. Davis, L. M. Rynkiewicz and R. E. Pottinger.—p. 1062.
Further Studies on Experimental Endometriosis. R. B. Scott, R. W. Te Linde and L. R. Wharton.—p. 1082.
Observations on Pathogenesis of Premature Separation of Normally Implanted Placenta. W. F. Mengert, J. H. Goodson, R. G. Campbell and D. M. Haynes.—p. 1104.
Review of 1,000 Maternal Deaths in a Rural State. R. A. Ross.—p. 1113.
Nonoperative Treatment of Stress Incontinence in Women. L. R. Wharton. —p. 1121.
Modern Treatment of Tuberculosis and the Gynecologist. E. M. Jameson. —p. 1131.
Influence of Antibiotic Therapy on Intrauterine Bacterial Flora. H. C. Hesseltine, J. Bustamante and M. Loth.—p. 1140.

Pregnancy and Diseases of Adrenal Glands.—This report is based on 18 pregnancies in 11 patients with disturbances of adrenal function seen at the Mayo Clinic during the period 1940-1952 and other such pregnancies reported in the literature. Distribution of pregnancies in the cases reported was as follows: seven in four patients with active Cushing's syndrome; two in five patients with adrenal cortical insufficiency associated with Addison's disease in three women, following surgery for Cushing's syndrome in one and following removal of bilateral pheochromocytomas in one; and four in four patients who had been treated for Cushing's syndrome and in whom no clinical adrenal cortical insufficiency was present. Hypertension is probably the chief danger to those patients with hyperfunction of the adrenal cortex or medulla. Fetal loss occurred in three of

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Titles marked with an asterisk (*) are abstracted.

the seven pregnancies associated with active Cushing's syndrome. Among the nine women with pheochromocytomas (two reported here and seven in the literature), the fetal loss was five. The hypertension of pheochromocytoma may be of serious maternal consequence, especially if the lesion is undiagnosed at deliverery; two of six patients reported in the literature died of shock postpartum. Three adrenal states may bring about "obstetric shock" with serious postpartum collapse and death: (1) postpartum crisis in women with adrenal cortical insufficiency; (2) postpartum collapse in parturients due to a pheochromocytoma; and (3) acute adrenal insufficiency after delivery in patients with adrenal cortical atrophy induced by previous cortisone therapy. This condition has not been seen post partum, but should be borne in mind as a possibility for every woman who has had cortisone therapy. Significant elevation during pregnancy of the values for urinary 17-ketosteroids and corticosteroids was found in two patients with adrenal cortical insufficiency; the source of this increase is unknown.

Toxemia of Pregnancy.—The nitrous oxide method for the quantitative determination of the cerebral blood flow has been used during the past six years to make approximately 600 cerebral blood flow investigations of 325 pregnant women. Values for mean arterial blood pressure, cerebral blood flow, cerebral oxygen metabolism, cerebral vascular resistance, respiratory quotient, amounts of oxygen and carbon dioxide in the veins and arteries, and arteriovenous oxygen difference were determined in normal pregnant and normal nonpregnant women. The arterial and venous concentrations of oxygen and carbon dioxide are much lower in pregnant women, apparently due to so-called hydremia of pregnancy with relative increase in plasma volume and lowered hemoglobin concentration; but normal pregnancy causes no alteration in the normal pattern of oxygen utilization by the brain. The values were established for preeclampsia, essential hypertension with superimposed acute toxemia, and eclampsia. The over-all cerebral blood flow is not affected by these three types of toxemia of pregnancy. Cerebral oxygen metabolism is normal in nonconvulsive toxemia but depressed during the coma of eclampsia, even though the arterial oxygen supply to the brain is normal, suggesting a histotoxic anoxia. The cerebral vascular resistance is increased in both convulsive and nonconvulsive toxemia; this is consistent with the conception of toxemia of pregnancy as generalized vasospasm. Two vasodilating drugs, veratrum viride and hydralazine (Apresoline) hydrochloride, have been evaluated in the treatment of toxemia of pregnancy. The former was administered to 20 women; 9 were normal prenatal patients and 11 had toxemia of pregnancy. There were no complications occasioned by administration of the drug, and the fetal heart tones were not adversely affected. After administration of veratrum viride there was a drop in the mean arterial blood pressure and the cerebral vascular resistance of all the women to subnormal levels. The other cerebral values were not significantly affected. Hydralazine was given to 13 toxemic patients and 9 normal pregnant women. There were some transient, but no permanent, ill-effects, and the fetal heart sounds remained normal. The drops in mean arterial blood pressure and cerebral vascular resistance were not so large as those following administration of veratrum viride. Cerebral blood flow and cerebral oxygen metabolism were increased by about the same amount in both the normal and the toxemic patients. It is suggested that cerebral vascular resistance is a more important factor in toxemia of pregnancy than systemic blood pressure and that blood viscosity, brain edema, and spinal fluid pressure play less important parts in the cerebral vascular resistance of toxemia of pregnancy than does augmented tone of the cerebral vessels. The cause of this increase is not known, but it must depend on neurogenic, chemical, or humoral influences. Since the two vasodilators studied do not affect either of the first two modes of influence, the last assumes the greatest importance. It has been recently discovered that the cerebrum secretes a vasopressor substance and that the brain possesses a chemoreceptor system that has an important place in determining the body's response to vasoactive substances; therefore, the possibility must be strongly considered that some humoral agent is the cause of the vasospasm of toxemia of pregnancy and is involved in the action of the hypotensive agents studied.

American Journal of Physiology, Washington

174:331-498 (Sept.) 1953. Partial Index

Effect of X-Irradiation upon Performance of Daily Exhaustive Exercise by Rat. D. J. Kimeldorf, D. C. Jones and T. J. Castanera.—p. 331.
Choline Studies in Rats Following Whole-Body X-Irradiation. D. J. Buchanan, W. J. Darby, E. B. Bridgforth and others.—p. 336.
Effect of Hemorrhage on Estimated Hepatic Blood Flow and Renal Blood Flow in Dogs. H. O. Heinemann, C. M. Smythe and P. A. Marks.—p. 352.
Liver and Muscle Glycogen as Influenced by Alloxan. O. S. Cullimore, P. J. Bradshaw, N. R. Blatherwick and E. F. Beach.—p. 357.
Electromyography in Normal, Dilated, Transected and Transplanted Ureter. R. Baker and J. Huffer.—p. 381.
Development of Experimental Renal Hypertension in Dog Following Reduction of Renal Artery Pulse Pressure Without Reducing Mean Pressure. E. W. Hawthorne, S. L. C. Perry and W. G. Pogue.—p. 393.
Renal Function During Arterial Hypertension Produced by Chronic Splanchnic Nerve Stimulation in Dog. W. G. Kubicek, F. J. Kottke, D. J. Laker and M. B. Visscher.—p. 397.
Chemical Changes in Rabbit Brain During Anoxia. H. G. Albaum, W. K. Noell and H. I. Chinn.—p. 408.
Hemodynamic Effects of Continuous Intravenous Infusion of Neo-Synephrine. D. W. Knapp, B. K. Hutt and S. M. Horvath.—p. 413.
Relationship Between Actions of Adrenocortical Steroids and Adrenomedullary Hormones in Production of Eosinopenia. W. L. Henry Jr., L. Oliner and E. R. Ramey.—p. 455.
Metabolism of Plasma Expanders Studied with Carbon-14-Labeled Dextran. I. Gray—p. 462.

175:1-170 (Oct.) 1953. Partial Index

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Effect of Hypothalamic Lesions on Adrenal Cortical Response to Stress in Rat. S. M. McCann.—p. 13.
Effect of X-Irradiation on Serum Potassium and Sodium Levels in Rabbits. R. D. Goodman and M. Vogel.—p. 29.
Renal Tubular Resistance to Mercurial Action in Sodium Deficiency. L. Rice, J. Frieden, B. M. Kaplan and M. Smith.—p. 45.
Peripheral Vascular Responses to Remote Thermal Burns and Frostbite as Influenced by Heparin and Paritol. B. J. Sullivan and W. K. Masterson.—p. 56.
Stress and Capillary Resistance (Capillary Fragility). J. Kramár.—p. 69.
Physiological Properties of Renin and Hypertension. H. Goldblatt, H. Lamfrom and E. Haas.—p. 75.
Bio-Electric Phenomena as Etiologic Factor in Intravascular Thrombosis. P. N. Sawyer and J. W. Pate.—p. 103.
*Salt-Fed Animals as Test Object for Pressor Substances in Blood of Hypertensive Animals. D. B. Gordon, D. R. Drury and S. Schapiro.—p. 123.
*Terminal Phenomena Associated with Massive Doses of X-Rays. H. L. Andrews and K. C. Brace.—p. 138.
Effects of Irradiation on Anabolism of Antibody and of Serum Albumin and Globulin. K. M. Stevens, I. Gray and M. S. Schwartz.—p. 141.
Effect of Whole Body X-Irradiation on Blood Constituents. W. E. Cornatzer, O. Engelstad and J. P. Davison.—p. 153.
Synergistic Effect of Internal Irradiation and Phlebotomy on Production of Cirrhosis and Ascites in Dogs. P. F. Hahn, E. L. Carothers and H. Brummit.—p. 162.

Pressor Substances in Blood.—The theory that various forms of hypertension may be due to the presence of a pressor substance in the blood is an appealing one, and many attempts have been made to verify it experimentally. The possibility was considered that renal hypertension may involve, in addition to the presence of a pressor substance in the blood, a sensitization of the animal to the pressor action of this substance. This idea seemed reasonable in view of numerous experimental results showing a heightened sensitivity of the renal hypertensive animal to various pressor agents. In view of evidence of an increased amount of sodium in various tissues of the renal hypertensive animal, it was further considered possible that the increased sensitivity of the hypertensive animal might be dependent on this change in its cellular composition. Cross transfusions were carried out between renal hypertensive rabbits and rabbits that had previously been drinking 2% sodium chloride solution for three or more weeks. There was an elevation of the arterial blood pressure of the animals fed salt in every instance after completion of cross transfusion and recovery of the animals from anesthesia. Two series of control experiments were carried out. In one, cross transfusions between renal hypertensive rabbits and normal rabbits were performed. There were no significant elevations of the normal rabbits' blood pressure after this procedure. In the other series, cross transfusions between normal rabbits and salt-fed rabbits were performed. In these experiments, no rise or a very small rise in the blood pressure of the salt-fed rabbits was observed. These results indicate that there is a

pressor substance in the blood of renal hypertensive rabbits. Normal rabbits do not react to this substance. Rabbits "densitized" by drinking a hypertonic solution react to this substance by an elevation of blood pressure. It is suggested that the hypertensive animal responds to the same substance because of an increased reactivity of its vascular system.

Terminal Phenomena Associated with Massive Irradiation.—Most studies on the biological effects of high energy radiation have been made with doses causing death in a matter of days. During this interval secondary effects and bacterial invasion may develop and obscure the primary radiation effects. The present studies were undertaken with the thought that a single dose of x-rays from 50 to 100 times that ordinarily used would emphasize the primary radiation effects and elicit previously overlooked phenomena. A series of 22 guinea pigs were exposed to 25,000 r of x-rays over the whole body or with various areas shielded. Measurements of rectal temperature, electrocardiograms, and single lead electroencephalograms were taken at intervals until the animals died or were killed for blood chemistry studies. All animals showed a striking drop in the heart rate and body temperature, and slow waves appeared in the electroencephalogram. Clinical signs of central nervous system dysfunction appeared after a few thousand roentgens had been delivered, and these persisted until death. The dysfunction appears to be subcortical and is not the direct result of ionization in the brain tissue.

Journal of Bacteriology, Baltimore

66:377-504 (Oct.) 1953. **Partial Index**

Explanation for Arithmetic Linear Growth of Mycobacteria. W. A. Volk and Q. N. Myrvik.—p. 386.
Relationship Between "Chloramphenicol Reductase Activity" and Chloramphenicol Resistance in Escherichia Coli. J. R. Merkel and E. Steers.—p. 389.
Auto-Adaptation: New Phenomenon Observed in Bacterial Population. B. E. Wright.—p. 407.
Effects of Some Salts on Germicidal Action of High Voltage Cathode Rays Towards Micrococcus Pyogenes Var. Aureus. C. G. Dunn.—p. 421.
Requirement of Electrolytes for Adsorption of Escherichia Coli Antigen by Red Blood Cells. E. Neter and N. J. Zalewski.—p. 424.
Production of Acid Phosphatase by Certain Species of Actinomyces. A. Howell Jr. and R. J. Fitzgerald.—p. 437.
Significance of Lactose Fermentation and Its Relationship to Resistance in Klebsiella Pneumoniae. V. J. Cabelli and M. J. Pickett.—p. 443.
Blood Media for Cultivation of Mycobacterium Tuberculosis: VII. Comparison of Blood Agar-Penicillin and Lowenstein-Jensen Media Under Routine Diagnostic Conditions. M. S. Tarshis, P. C. Kinsella and M. V. Parker.—p. 448.

Journal of Thoracic Surgery, St. Louis

26:441-550 (Nov.) 1953

Present Chaos Regarding Resection of Residual Caseous Lesions in Pulmonary Tuberculosis. E. J. O'Brien, A. C. Miller, L. Linda, P. T. Chapman, K. Koster and P. V. O'Rourke.—p. 441.
*Use of Isoniazid as Prophylactic Antibacterial Agent in Pulmonary Resection for Tuberculosis. M. E. Childress, M. E. Thoren and A. C. Daniels.—p. 447.
Influence of Long-Term Chemotherapy on Surgery of Pulmonary Tuberculosis. J. D. Steele, B. G. Narodick and A. V. Cadden.—p. 459.
*Segmental Resection for Pulmonary Tuberculosis (300 Cases). J. M. Chamberlain, C. F. Storey, R. Klopstock and C. F. Daniels.—p. 471.
Electrolyte Studies in Pericardial Resection. W. B. Crandell, A. Yeomans, D. L. Hoffman and G. H. Stueck.—p. 486.
Bronchial Closure in Pulmonary Resection: Clinical and Experimental Study Using Pedicled Pericardial Fat Graft Reinforcement. L. A. Brewer III, E. L. King, L. J. Lilly and A. F. Bai.—p. 507.
Circulation Through Atelectatic Lung in Man. V. O. Björk.—p. 533.
Resection of Multiple Pulmonary Metastases 14 Years After Amputation for Osteochondrogenic Sarcoma of Tibia. E. P. Mannix Jr.—p. 544.

Isoniazid in Pulmonary Resection.—Twenty-three patients undergoing pulmonary resection for tuberculosis were given isoniazid as a prophylactic antibacterial agent starting 48 hours before operation and continuing for two months after. These patients had not been previously treated with isoniazid but had in most cases received streptomycin and/or *p*-aminosalicylic acid. The tubercle bacilli of eight were resistant to streptomycin. There were no postoperative complications attributable to tuberculosis, though there were five complications from other causes. Isoniazid affords adequate protection from the tubercle bacillus during the operative period and for a minimum of two months after operation. Unfortunately, patients who have not already been treated with isoniazid are becoming increasingly rare; it is suggested that isoniazid be withheld from patients for whom pulmonary resection may be indicated in the future.

Segmental Pulmonary Resection.—A series of 300 consecutive patients who underwent segmental resection for pulmonary tuberculosis was followed for from one to five years. At the time this paper was written, 93.7% were alive and well, 3.3% were living but still had the disease, and 3% were dead. The indication for segmental or subsegmental resection is a fibrocaseous lesion with or without cavitation confined to one or two segments of a lobe, but still large enough that clinical opinion is doubtful of the patient's future security. The preoperative preparation is the same as for any major operation, with special attention to laminography and especially the lateral laminography. An anatomic segmental resection is done for moderate and far-advanced lesions. For minimal lesions, a wedge resection or local enucleation is possible. Subsegmental resections may also be done anatomically. Tuberculosis is usually bilateral though not bilaterally active. Only the major active focus is removed by segmental resection. The importance of other foci is dependent on the size and location. Anterior foci are less hazardous than posterior ones. The problem of overdistention is not great after segmental resection, but obliteration of the dead space is essential to success. Occasionally a periosteal plombage procedure without resection of the ribs is done for this reason. In obliterative pleuritis mobilization of the lung is not done when preoperative fluoroscopy reveals a good diaphragmatic movement. The functional loss is less if a small tailoring surgical procedure is done in the thoracic vault. Postoperative care must be vigilant. Patent airways (intratracheal suction), the control of pain, the use of antibiotics, the obliteration of dead space by complete reexpansion of the lung using active suction and checked by frequent postoperative x-ray examinations are important to a successful result.

Maryland State Medical Journal, Baltimore

2:527-572 (Oct.) 1953

Interrelations of Thyroid and Iodine Metabolism. T. S. Danowski.—p. 530.
*Excision of Aortic Aneurysms. H. T. Bahnson.—p. 537.
*Indications for and Results of Surgery for Mitral and Aortic Stenosis. R. P. Glover.—p. 547.
Non-Operative Treatment of Stress Incontinence in Women. L. R. Wharton.—p. 555.

Excision of Aortic Aneurysms.—Between January, 1952, and March, 1953, the author and his colleagues operated on 20 patients with aortic aneurysms with the intention of excising the aneurysms. Five of these patients died during or shortly after the operation. Of the 15 patients for whom the operation was successful, 3 were still in the hospital at the time this paper was written. The other 12 were well and essentially symptom-free. The author feels that excision is a better form of treatment for aneurysms in all sections of the aorta than ligation, wiring, so-called "cellophane wrapping," or endoaneurysmorraphy.

Surgery for Mitral and Aortic Stenosis.—Modern techniques of cardiac surgery have made the treatment of mitral and aortic stenoses possible by using the Glover-Avery guillotine and an aortic dilating instrument, respectively. Since surgical treatment is more beneficial to the patients in the early stages of these disorders than to those in the later stages, an increased responsibility rests with the physician to select these patients before their disease becomes so advanced that it presents dangerous complications or contraindications for surgery. Five hundred mitral commissurotomies have been performed by the author and his colleague. Of the first 164 patients surgically treated, 49% were restored to excellent health, 27% were improved in various degrees, 12% were unimproved, 6% died in connection with the operation, and 5% died later in the course of a one to four year follow-up. Twenty-two aortic commissurotomies were performed, six of which were combined with mitral commissurotomies because of bivalvular stenosis. Of these patients, four were restored to excellent health, nine

were improved (including three with double commissurotomies), two were unimproved, and seven died (including three others with double procedures.) It is important to note that in both operative series no patients in the asymptomatic stage were operated on, and all patients in the second stage of the two diseases, when symptoms first began to appear, showed good results of surgery. The results became progressively poorer in the three later stages of stenosis with complications.

Mental Hygiene, Albany, N. Y.

37:529-712 (Oct.) 1953. **Partial Index**

Mental Health: Local Public-Health Responsibility. H. E. Jensen.—p. 530.
Progressive Industry and the Worried Employee. J. S. Felton.—p. 545.
Aspects of Mental Health in Teaching and Learning. P. Blos.—p. 555.
Maladjustment and Maternal Rejection in Retrolental Fibroplasia. H. Barry Jr. and F. E. Marshall.—p. 570.
How the Child Reacts to His Physical Defects. E. Podolsky.—p. 581.
Consultation Service to Public Schools by Mental-Health Team. K. J. Perkins.—p. 585.
Dynamics of Marital Relationship. A. Stone and L. Levine.—p. 606.
Halfway House: Role of Laymen's Organizations in Rehabilitation of the Mentally Ill. L. E. Reik.—p. 615.
Rates of Discharge and Rates of Mortality Among First Admissions to New York Civil State Hospitals: Third Paper. B. Malzberg.—p. 619.

Pediatrics, Springfield, Ill.

12:341-470 (Oct.) 1953

Maximum Breathing Capacity and Vital Capacity in Female Children and Adolescents. B. G. Ferris Jr. and C. W. Smith.—p. 341.
Total Amino Acid Composition in Mature Human Milk: Analysis by Ion Exchange Resin Column Chromatographic Technic. M. W. Cheung, E. L. Pratt and D. I. Fowler.—p. 353.
*Hypertensive Phase of Acute Nephritis: Specific Therapy with Derivative of Veratrum Viride. S. W. Royce.—p. 358.
*Syndrome of Congenital Hemihypertrophy, Shortness of Stature, and Elevated Urinary Gonadotropins. H. K. Silver, W. Kiyasu, J. George and W. C. Deamer.—p. 368.
Association of Escherichia Coli Serogroup 0111 with Two Hospital Outbreaks of Epidemic Diarrhea of Newborn Infant in New York State During 1947. E. Neter, R. F. Korns and R. E. Trussell.—p. 377.
Chronic Familial Nonspherocytic Hemolytic Anemia. E. L. Lipton, H. J. Grossman and J. B. Richmond.—p. 384.
Studies of Immunology of Newborn Infant: V. Effect of Dietary Protein on Antibody Production. J. Dancis, J. J. Osborn and J. F. Julia. —p. 395.
Diabetes Insipidus in Infancy Resistant to Pitressin. M. Yun-Chen Kao and M. M. Steiner.—p. 400.
Growth in Head Width During First 12 Years of Life. H. V. Meredith. —p. 411.
Payment for Hospital Care of Premature Infants. H. M. Wallace, L. Baumgartner, M. A. Losty and H. Rich.—p. 430.

Hypertensive Phase of Acute Nephritis.—Alkavervir (Veriloid) was given to five boys and one girl between the ages of 4 and 13 who were in the crucial hypertensive phase of acute nephritis that is difficult to control. The drug was administered intravenously in two phases: an initial, fairly rapid infusion of 0.022 cc. of alkavervir solution (diluted to a volume of 10 cc. with 5% dextrose solution) per kilogram of body weight, followed by a slow intravenous drip of 0.13 cc. of alkavervir solution per kilogram of body weight until a total of 22 cc. of the alkavervir solution per kilogram of body weight has been given at a speed of 5 drops per each 10 kg. of body weight and per minute. Prophylaxis of myocardial and cerebral damage resulting from a rapid rise of blood pressure is the main objective of treatment during the early phase of acute nephritis in children. Prompt remission of hypertension occurred in all patients after the administration of alkavervir. There was no evidence of toxicity of the drug and no undersirable side-effects other than mild nausea or vomiting were observed. The renal and cardiac status did not appear to be affected unfavorably by the treatment. The patients were followed for at least two years after the onset of their illness and remained well and normotensive. Although no ideal hypotensive agent is available at present, alkavervir deserves a therapeutic trial in children on a larger scale because of its capacity to reduce the peripheral resistance, to lower the blood pressure rapidly, and to maintain the blood flow and the cardiac output at normal levels.

Congenital Hemihypertrophy.—A syndrome of hemihypertrophy, shortness of stature, and elevated levels of urinary gonadotropins is described in a girl 5½ and a boy 6. In both of the patients the dwarfing and the asymmetry that involved one-half of the body had been present since birth, and elevated gonadotropins for the age of the patients were observed in both. Courses of thyroid extract were tried in both with no apparent acceleration of the rate of growth or gain in weight. Variations in urinary gonadotropins occur in ovarian or testicular agenesis in childhood, and shortness of stature and congenital abnormalities may be part of the clinical picture of this condition. The absence of the commoner congenital defects of that syndrome, such as webbing of the neck, cubitus valgus, coarctation of the aorta, and the presence of a normal uterus and tubes on rectal examination of the girl, and normal testes on palpation in the boy made the diagnosis of a variant of ovarian (testicular) agenesis unlikely. The syndrome described in the authors' patients and the syndrome of ovarian agenesis may be related conditions. A café-au-lait area of the anterior abdominal wall was observed in the girl, and, therefore, neurofibromatosis with involvement of the hypothalamic or pituitary region must be considered as a possible cause of the disturbance. No other manifestation of neurofibromatosis was observed in the girl, and no evidence of the disease was found in the boy with the same clinical syndrome as the girl. Although numerous concepts of the etiology have been suggested in the literature, the origin of congenital hypertrophy has not yet been satisfactorily explained.

West Virginia Medical Journal, Charleston

49:279-310 (Oct.) 1953

*Urinary Cloaca Fashioned from Short Distal Segment of Loop Colostomy: (Preliminary Case Report). T. P. Mantz and K. Kasti.—p. 279.
Radioisotopes in Clinical Medicine. W. K. Yates.—p. 282.
Duodenal Diverticula. J. C. Condry.—p. 286.
Fibromyomata of Uterus in Pregnancy. C. S. Flynn.—p. 288.
Chloral Hydrate: A Resumé. F. J. Moore.—p. 292.
Management of Common Allergic Diseases. M. Koenigsberg.—p. 295.

49:311-338 (Nov.) 1953

Carcinoma of Breast (Including Carcinoma in Situ) and Its Grades of Malignancy and Prognosis. A. C. Broders Sr.—p. 311.
Ectromelia: Case Presentation: (Final Report). J. C. Arnett.—p. 316.
Problems Facing West Virginia Hospitals in Providing Care for the Aged. W. R. Huff.—p. 318.
Closure of Tympanic Membrane Perforations by Chemical Cautery. D. W. Mullins.—p. 321.
Tumors of the Neck. L. H. Clerf.—p. 324.

Urinary Cloaca Formed from Segment of Loop Colostomy.—Ureterosigmoidostomy carries a high morbidity and mortality rate, and long survival is the exception. Hydronephrosis and pyelonephritis almost inevitably develop and are responsible for the high mortality. The use of isolated intestinal segments as bladder substitutes has greatly reduced the early and latent kidney complications that inevitably develop when ureterointestinal anastomosis is made into the intact bowel; however, an accessory abdominal opening is necessary. In the case presented, Mantz and Kastl transplanted the ureters into a more satisfactory urinary receptacle, namely into a distal segment of a loop colostomy. This bowel segment is independent of the fecal stream. The chance of retrograde infection is less. There is no obstruction to the urinary outflow. Because of these advantages and the simplicity of this technique, pelvic exenteration could become more widely used. Selected cases of postirradiation recurrent cancer of the cervix might be surgically treated more frequently if the late complications of kidney infection, ureteral obstruction, and electrolyte disturbance could be prevented. In the first stage of this procedure, a loop colostomy is done. At a later date, the ureters are transplanted into the short distal segment of the loop colostomy; this is followed by pelvic exenteration. The described method insures an unobstructed flow of urine, without back pressure and direct contamination of the ureteral orifice. This common opening on the abdominal wall seems to be a most desirable method and works very well with a Rutzen bag.

FOREIGN

Archives of Disease in Childhood, London

28:343-422 (Oct.) 1953

Hepatic Cirrhosis Associated with Fibrocystic Disease of Pancreas: Clinical and Pathological Reports of Five Patients. R. Webster and H. Williams.—p. 343.

*Congenital Hypertrophic Pyloric Stenosis in Twins. J. D. Metrakos.—p. 351.

Case of Fanconi's Syndrome with Study of Peroxidase Activity of Erythron. O. Higashi, E. Koseki and M. Higuchi.—p. 359.

Erythrogenesis Imperfecta or Congenital Hypoplastic Anaemia (Diamond-Blackfan Type). O. D. Fisher and F. M. B. Allen.—p. 363.

Favism in London. J. H. Diggle.—p. 369.

A.C.T.H. in Nephrosis. G. C. Arneil and H. E. C. Wilson.—p. 372.

*Response of Acute Anterior Poliomyelitis to Adrenergic Blocking Agents and Hot Pack Therapy. W. W. Whitten and J. G. Cramer.—p. 381.

Observations on Pyuria in Children. J. M. Stansfeld and J. K. G. Webb.—p. 386.

Classification of Enuresis. E. M. Poulton and E. Hinden.—p. 392.

Foetal Ascites. J. M. Lord.—p. 398.

Urticaria Neonatorum (Erythema Toxicum Neonatorum). H. V. L. Finlay and J. P. Bound.—p. 404.

Investigation into Certain Aspects of Pertussis. C. W. Kesson and M. Harling.—p. 409.

Skin Thrush in Early Infancy. A. Holzel.—p. 412.

Pyloric Stenosis in Twins.—The occurrence of pyloric stenosis in twins is presented. In four pairs of monozygous twins, both twins were affected in three pairs and one twin was affected in the fourth pair. In one dizygous pair, only one twin was affected. These five pairs are combined with 127 pairs reported in the literature, and an analysis is made employing the "twin method." For the final analysis only those cases were accepted in which the hypertrophic pylorus was identified unequivocally at operation or at necropsy and in which there was ample evidence to support either monozygous or dizygous origin. Of the 132 pairs, only 47 satisfied these conditions. Of these, 18 were monozygous and 29 dizygous. In 12 (66.7%) of the monozygous pairs, both twins had the condition, whereas in only 1 (3.49%) of the 29 dizygous pairs both twins had the condition. From a study of cases in twins, therefore, there seems to be little doubt that there is a hereditary factor involved in the etiology of hypertrophic pyloric stenosis. It also appears that some prenatal or postnatal factor may be necessary for the development of the hypertrophy and that such an environmental factor may be effective in only one of a pair of twins even when the twins are genetically alike.

Acute Anterior Poliomyelitis.—The effects of tolazoline (Priscoline) hydrochloride, Dibenamine hydrochloride (N,N-dibenzyl-β-chloroethylamine), and hot packs on muscle spasm and pain of patients with acute anterior poliomyelitis were studied. Although the number of patients studied was too small for a significant statistical comparison of the results of all forms of therapy, the following conclusions were made. Relief of severe subjective pain at rest is practically always accomplished within one-half to 8 hours after administration of tolazoline or Dibenamine, whereas packs are much less effective and take considerably longer. Relaxation of muscle spasm with drugs is either quite definite or completely absent, suggesting that more than vasospasm may be etiologically involved. At least 50% of the patients responded favorably enough to eliminate the need for hot pack therapy. Although actual limitation of mobility might still remain after the administration of drugs in some cases, stretch pain in muscle was frequently eliminated, permitting early physical therapy. In some cases, initial improvement after receiving drugs was superseded by severer spasm in five or six days. In those cases tested with Dibenamine, this was found to result from the use of the oral route which is ineffective. Early active and passive motion within the limits of pain must be instituted along with drug therapy for favorable results. No severe toxicity was observed with administration of either drug. In general, drug therapy is worthy of trial in most cases of poliomyelitis as the presence or lack of response will soon be evident; early use of hot packs, if necessary, will not be delayed. In this study, the muscles in patients with the highest temperature were in spasm, followed by weak and then by normal muscles. Response to therapy in terms of muscle temperature was greatest in weak muscles. No abnormally low skin temperatures were observed, and the response to Dibenamine was more marked than to tolazoline. In terms of muscle temperature, the effective therapeutic response was greatest when administration of Dibenamine was followed by that of tolazoline and hot packs. No obvious absolute muscle ischemia was noted, but it is suggested that increased tissue metabolism may create a relative anoxia that may be relieved by an increase in blood flow. Hypersensitivity of denervated muscles to acetylcholine may be enhanced by adrenolytic agents and may, therefore, explain the marked temperature increase following therapy. Further studies on tissue metabolism and blood flow are indicated before an adequate explanation will be available for muscle spasm and pain.

Archivum Chirurgicum Neerlandicum, Arnhem

5:169-252 (No. 3) 1953. **Partial Index**

*Renal Carbuncle. E. A. Immink.—p. 174.

*What Determines the Incidence of Acute Appendicitis (Statistical Observations). A. Grassberger.—p. 180.

Method for Treatment of Low Ureteral Strictures by Means of Catheter-Led Flexible Dilator. H. P. Vlietstra.—p. 197.

Treacher-Collins Syndrome (Mandibulo-Facial Dysostosis). A. Barendregt.—p. 209.

*Present Status and Future of Resection Therapy in Pulmonary Tuberculosis. (Based on Experiences of 790 Resections in 1943-1953). L. D. Eerland.—p. 213.

Transplantation of Formaldehyde-Preserved Human Aortic Graft in Case of Accidental Injury of Abdominal Aorta. M. W. Van Weel.—p. 233.

Renal Carbuncle.—Immink presents the histories of three patients with renal carbuncle. All three were treated with penicillin. In two of them, a boy, aged 9, and a woman, aged 36, treatment with penicillin was followed by cure, but in the third patient, a man, aged 52, pyrexia persisted despite treatment with large doses of penicillin. Four weeks after admission, the right kidney was exposed. An inflammatory growth the size of a swan's egg was found, with a thick, inflamed wall. Three liters of cream-colored, odorless pus were removed by puncture, after which the large sac collapsed. A small, apparently normal, part of the kidney was found on its under surface. The inflammatory process appeared to have originated from the upper pole and completely destroyed the remainder of the kidney. The process extended into the true pelvis. Since partial nephrectomy would probably have given rise to disturbances in wound healing, the right kidney was completely extirpated. The postoperative course was uneventful. The microscopic study of the surgical specimen disclosed an abscess membrane originating from a renal carbuncle. The author believes that spontaneous recovery from renal carbuncle is rare. Only intravenous pyelography affords a means of verification and of preventing a sudden flare-up of an apparently spontaneously healed process. The dose of penicillin should be increased if there is no response, but absorption is no longer possible once an abscess has formed. It is possible, however, to obtain sterilization of the pus by penicillin treatment. The duration of treatment is determined by the erythrocyte sedimentation rate and the leukocyte count. It may be said that penicillin treatment has reduced the number of surgical cases from 80 to 40%.

Incidence of Appendicitis.—This report is based on a total of 3,229 cases of acute appendicitis that were observed during the period from 1932 to 1950. The sex distribution was about even. During the years of poor nutrition, the incidence of acute appendicitis decreased greatly. One-fourth of all cases occurred in children under 14, another fourth in those between 15 and 18 years, a third fourth in persons between 19 and 28, and the last fourth in persons 29 and over. There exists a predisposition in youth and in women up to the menopause. The mortality of acute appendicitis was 1.52%. Perforation existed on admission in 10.52%; 13.87% of these died. Of the patients with perforation, 29% were less than 5 years old; the percentage decreased with increasing age, but increased again after the age of 40. Perforation is more frequent in men than in women, but men survive perforation oftener than women. The author

believes that the incidence of appendicitis can be reduced by avoiding excessive intake of fat and meat and by providing an adequate amount of roughage in the diet, particularly in the age groups that seem predisposed.

Resection in Pulmonary Tuberculosis.—Eerland says that a total of 790 pulmonary resections for tuberculosis were carried out by him and Dr. Oterdoom since 1943. This review is concerned chiefly with 745 patients operated on before June, 1953, and of whom 204 were subjected to pneumonectomy, 217 to lobectomy, and 324 to segmental resection sometimes combined with lobectomy. There were 16 surgical deaths (2.1% of 745), and 17 died later (15 of tuberculosis). Of the first 249 patients, who had been operated on more than two years ago, 224 are still alive, and 87% of them lead a normal active life. The sputum has become negative in the same percentage of cases. The results in 167 patients who were subjected to segmental resection and who had been observed for from eight months to three years are encouraging. There were no deaths, and the functional results were good. Of the 162 patients, 147 had negative sputum and 106 again led an active life; 22 were working half time. In 15 patients, resection had to be repeated. A bilateral resection was successfully performed in 16 patients. Emphasis is placed on the fact that pulmonary resection is only part of the therapy. After the operation the patients still require a rest cure in a sanatorium. The author believes that the efficient organization of the combat of tuberculosis in Holland is largely responsible for the good results obtained with resection. From the onset, the patients are always under the care of specialists in tuberculosis.

British Journal of Ophthalmology, London

37:641-704 (Nov.) 1953. **Partial Index**

Bulbar Pressure Test in Glaucoma. P. L. Blaxter.—p. 641.
Cataracts in Galactosaemia. F. D. McAuley.—p. 655.
Definition of Visual Acuity. M. Gilbert.—p. 661.
Anterior Flap Sclerotomy with Basal Iridencleisis. H. B. Stallard.—p. 680.
Congenital Toxoplasmosis. J. Hill.—p. 689.

British Journal of Surgery, Bristol

41:113-224 (Sept.) 1953

*Gastrojejunocolic Fistula. A. G. R. Lowdon.—p 113.
Colo-Colic Intussusception in Adult. D. H. Teasdale.—p. 128.
V-Y Correction for Varus Deformity of Fifth Toe. J. N. Wilson.—p. 133.
Adenocarcinoma of Appendix. S. G. Nardell.—p. 136.
Solitary Pyogenic Abscess of Liver Treated by Closed Aspiration and Antibiotics: Report of 14 Consecutive Cases with Recovery. A. J. S. McFadzean, K. P. S. Chang and C. C. Wong.—p. 141.
Recurrent Perforation of Jejunum Due to Lymphosarcoma. H. M. Bradmore and C. G. Scorer.—p. 152.
Cholecystitis Glandularis and Diverticula of Gall-Bladder. E. S. J. King. —p. 156.
*Results of Vagotomy in Treatment of Peptic Ulcer. B. H. Hand and D. H. Patey.—p. 161.
Molluscum Sebaceum. J. M. Beare.—p. 167.
Roux Loop. P. R. Allison and L. Tavares da Silva.—p. 173.
An Unusual Combination of Aberrant Hepatic Arteries. C. P. Mills. —p. 181.
Scrotal Cystoceles: Report of Case with Papillomata. R. Marcus.—p. 182.
Meconium Ileus: Report of Successfully Treated Case and Study of Surgical Aspects of Series of 14 Cases. H. H. Nixon.—p. 184.
Relief of Pain in Chronic Pancreatitis by Sympathectomy. D. M. Morrissey.—p. 189.

Gastrojejunocolic Fistula.—A fistulous communication between the transverse colon and the stomach or jejunum occurred in 40 men and 1 woman after gastroenterostomy performed for duodenal ulcer and occasionally for gastric ulcer. Four of the 41 patients had recurrence of fistula after surgical treatment, and in 1 of these a second recurrence occurred. Thus 46 separate fistulas are included in the author's report. The types of anastomosis that preceded these 46 fistulas were posterior gastroenterostomy in 39, anterior (antecolic) gastroenterostomy in 2, partial gastrectomy in 4 (2 with retrocolic and 2 with antecolic anastomoses), and Devine's pyloric exclusion operation in 1. The interval between these previous operations and the onset of symptoms of fistula varied from 1 month to 21 years. The triad of symptoms that is conclusive evidence of a fistula includes diarrhea, fecal vomiting, and loss of weight. In 3 of the 41 patients, however, there were no symptoms attributable to the fistula except occasional lower abdominal pain. Diarrhea, which was associated with 41 of the 46 fistulas, results from irritation of the small intestine by colonic content and is not attributable to gastric contents entering the colon through the fistula. Barium meal examinations were carried out in 22 patients and in 14 of these the fistula was not demonstrated; 32 patients were studied by barium enema and the fistula was demonstrated in 31 of these. The diagnosis thus can be confirmed by barium enema, while a barium meal may fail to demonstrate even a large fistula. The severity of the disease is variable. Only five patients had mild symptoms and were in good general condition, but most had severe diarrhea, and nutritional deficiencies rapidly developed that made them poor surgical risk and required relief as far as possible by preoperative parenteral therapy. The only effective treatment is surgical. Operation, in addition to removing the fistula, should include measures to relieve the ulcer diathesis. The few patients whose general condition is good and in whom the region of the fistula is not inflamed may justifiably be treated by excision of the fistula and partial gastrectomy in one stage, but most patients with this complication are not fit for an operation of such magnitude and should be treated by an operation in stages. The first stage should be a proximal transverse colostomy: this minor intervention is followed by relief of diarrhea and rapid improvement of general health. The definitive operation is performed two or three months later and should include resection of at least four-fifths of the stomach. The colostomy is closed two or three weeks later. The patient should be seen at regular intervals after this treatment, and if evidence of recurrent gastrojejunal ulcer is found a transthoracic vagotomy should be performed immediately. If a fistula occurs after adequate partial gastrectomy, a left thoracoabdominal exposure permits excision of the fistula and thoracic vagotomy.

Vagotomy in Treatment of Peptic Ulcer.—Between 1946 and 1950, vagotomy, either alone or, more commonly, combined with some other operative procedure, was performed in 60 men and 12 women with 70 duodenal ulcers and 5 jejunal ulcers. Of the 75 ulcers, 9 were treated by transthoracic vagotomy, 8 by transabdominal vagotomy, 32 by transabdominal vagotomy combined with pyloroduodenectomy, 9 by transabdominal vagotomy combined with gastrojejunostomy, and 17 by transabdominal vagotomy combined with gastrectomy. Two of the 72 patients died postoperatively, a mortality rate of 2.8%; one additional patient died after operation for gastric ulcer that recurred as a complication of the first operation; the total mortality rate thus was 4.2%. Gastric ulcer is a complication of vagotomy for duodenal ulcer, particularly of simple vagotomy (3 of 12 patients). It is less common in the combined procedures. The duodenal ulcer recurred in 5 of 12 cases of simple vagotomy and in 4 of 28 cases of vagotomy combined with pyloroduodenectomy. Ulceration also recurred in one out of eight cases of vagotomy combined with gastroenterostomy. There was no recurrence in 17 cases of vagotomy combined with hemigastrectomy, but follow-up of these cases was only for from one to two years. Vagotomy resulted in healing of all five gastrojejunal ulcers, but a gastric ulcer developed in one case in which vagotomy had been combined with gastroenterostomy. The immediate postoperative course of the patients with vagotomy was frequently anxious and stormy because of the pronounced gastric and duodenal atony. The abdomen may have to be reopened, and the atony may even result in death. Analysis of the postoperative results in relation to the barium meal findings showed no close relation with these findings, but some relation with the findings of the test meal was observed. A late postoperative morbidity in the patients with successful vagotomy was comparable with, although less severe than, that observed after subtotal gastrectomy. As with subtotal gastrectomy, these late symptoms are usually minor in their upsetting effect when compared with the original ulcer symptoms. The authors' present opinion is that the only group of cases of peptic ulcer in which vagotomy has an established position as a routine procedure is that of gastrojejunal ulcer after subtotal gastrectomy.

British Medical Journal, London

2:951-1004 (Oct. 31) 1953

Place of Forceps in Present-Day Obstetrics. T. N. A. Jeffcoate.—p. 951.
"Failed Forceps": Review of 37 Cases. R. G. Law.—p. 955.
Sickle-Cell Anaemia in Accra District of Gold Coast: Review of 20 Cases. G. M. Edington.—p. 957.
Health Visitor and Family Doctor. L. Roberts.—p. 962.
General Practitioner and Health Visitor. J. S. Thomas.—p. 964.
Hydrocortone and Soft-Tissue Lesions. J. Cyriax and O. Troisier.—p. 966.
Technique and Value of Aortography: Report of Case of External Iliac Arterial Graft. R. E. Horton and E. M. Nanson.—p. 969.
*Value of Single-Sample Test in Differential Diagnosis of Jaundice. I. Wang.—p. 971.
Fluid Content of Nucleus Pulposus as Factor in Disk Syndrome: Preliminary Report. A. Naylor and D. L. Smare.—p. 975.

Differential Diagnosis of Jaundice.—In cases of jaundice in which the clinical picture is problematical, biochemical tests, by suggesting an earlier solution, can be of value. On the one hand, the dangers of an unnecessary exploratory operation in a case of hepatogenous jaundice must be avoided, and, on the other, the patient must be guarded from the harmful effects of prolonged extrahepatic obstruction. The "single-sample tests" have the advantages of simplicity in obtaining the specimen with minimal discomfort to the patient, along with economy in time to the patient and the physician. However, many such single sample tests involve a number of biochemical determinations employing a wide range of tests. Bearing in mind that this is a clinical problem, the simpler tests, which can be carried out in any hospital laboratory, must be given prior consideration. The author investigated a series of blood tests regarding the value of these determinations for inclusion in a single sample test for the differential diagnosis of jaundice. Of the tests employed, the serum alkaline phosphatase activity, serum total cholesterol, and thymol turbidity were found to be of most significance. Determination of the plasma protein fractions was of no value and should be omitted from any single sample test. Tests for urobilinogen and urobilin in the urine were of value as supplementary information where the results of the blood tests were equivocal. Critical levels in the blood tests for the differentiation of hepatogenous from obstructive jaundice are suggested. Applying these criteria in a series of 104 cases of jaundice, the correct diagnosis of the type of jaundice was made in 91 cases. Of the remaining 13 cases, two gave equivocal results, while in 11 the biochemical findings were frankly misleading.

Bull. World Health Organ., Geneva

9:457-706 (No. 4) 1953. **Partial Index**

Plague Studies: 10. Control and Prevention. R. Pollitzer.—p. 457.
Epidemiological Investigations on Q Fever in Turkey. S. Payzin.—p. 553.
Immunological Classification of 25 European Strains of Poliomyelitis Virus. J. D. Verlinde, B. Hofman and E. Nihoul.—p. 559.
Stable Suspension for Vi-Agglutination Tests. K. Ando and H. Shimojo. —p. 575.

Deutsche medizinische Wochenschrift, Stuttgart

78:1489-1524 (Oct. 30) 1953. **Partial Index**

Campaign Against Cancer. H. Hellner.—p. 1489.
Tapeworm Cures (Taenia Saginata). R. Wigand and W. Warnecke. —p. 1493.
*New Form of Corticotropin Therapy in Rheumatic Polyarthritis. R. Prosiegel, A. Goelkel and U. Fuchs.—p. 1494.
Prophylaxis of Tetanus by Active Immunization. R. Hasche-Klünder and M. Pichert.—p. 1499.
Appendicitis in Nursing Infants and Young Children. H.-J. Wissfeld. —p. 1502.

New Form of Corticotropin Therapy in Rheumatic Polyarthritis.—The new form of corticotropin therapy discussed by Prosiegel and associates represents a modification of the cortisone-insulin therapy recommended by Henderson and others in 1951 and of Gordon's recommendation for infusing corticotropin by means of an intravenous drip. It was hoped that the addition of insulin would reduce the effective dose of corticotropin and thus make possible the continuation of corticotropin therapy over a longer period. The treatment was tried on 11 patients with chronic polyarthritis who were hospitalized throughout their treatment. During a preliminary period of eight days the clinical symptoms of the patients were estimated and at the same time their basic corticoid values were established. Some of the patients showed typical deformities, stiffening, and contractures and all had severe pains. The polyarthritis had existed for from 2 to 20 years. During the first five days of treatment, the patients were given daily 20 units of a corticotropin preparation by intravenous drip in from 400 to 800 cc. of Ringer's solution, over a period from six to eight hours. In the majority of patients, the pain subsided during these five days. In two cases in which this did not occur, this treatment was continued for another five days. During the second period in which the therapeutic effects were to be stabilized and further improved, the patients were given daily 8 units of corticotropin together with insulin, again in the form of an intravenous drip. Treatment was always interrupted during the last two days of the week. The insulin sensitivity of each patient varied greatly. Usually the initial dose of insulin was 8 units and this amount was increased by 2 units each day, but the daily dose never exceeded 20 units of insulin. Care was taken that no hypoglycemic manifestations occurred. In order to keep symptoms such as sweating and tremor in abeyance many of the patients were given 50 gm. of dextrose per day in the infusion fluid, during the last few weeks of the treatment. The results of treatment were quite satisfactory and corresponded on the whole to those obtainable with larger intramuscularly administered doses of corticotropin. Maintenance of the therapeutic effects required continued administration of small doses of corticotropin. The elimination of corticoids in the urine was generally high and did not decrease until therapy was interrupted. When only insulin was administered, or only small doses of corticotropin without insulin, the corticoid values in the urine were not so high. The authors discuss the possibility that insulin increases the synthesis of biologically active hormones of the adrenals.

Journal of Physiology, Cambridge

122:1-208 (Oct.) 1953. **Partial Index**

Kinetics of Penetration of Blood-Aqueous Barrier. H. Davson and P. A. Matchett.—p. 11.
Coagulation in Lymph. P. Fantl and J. F. Nelson.—p. 33.
Activity of Human Sweat Glands During Exposure to Cold. E. M. Glaser and T. S. Lee.—p. 59.
*Changes in Alveolar Carbon Dioxide Tension by Night and During Sleep. J. N. Mills.—p. 66.
Failure of Kidney to Respond to Respiratory Acidosis. D. Longson and J. N. Mills.—p. 81.
Muscle Spindles in Intrinsic Muscles of Human Tongue. S. Cooper. —p. 193.
Effect of Sodium Fluoride on Gastric Acid Secretion. J. Y. Bowie, G. Darlow and M. M. Murray.—p. 203.

Alveolar Carbon Dioxide Tension: Diurnal Rhythm.—Mills says that in the course of other work he together with Longson and Stanbury made many series of alveolar carbon dioxide determinations on subjects either fasting or with minimal hourly food intake, sometimes for 24 hours or more, and these provide data for consideration of diurnal rhythms. These were supplemented with determinations during sleep, either throughout the night or during short afternoon naps. Alveolar sampling during sleep presents difficulties, and since no technique is quite unobjectionable, several methods have been used. The studies reported here revealed that alveolar carbon dioxide tension sometimes shows a diurnal rhythm, with high values during the night even in subjects who remain awake fasting in constant conditions of rest or light activity. Alveolar samples collected by suction or by an end-tidal sampler during nocturnal sleep show a rise of tension no greater than that shown during sleepless nights. During afternoon sleep, assisted by hexobarbitone, the tension does not rise. Some of the Haldane-Priestley samples collected from a subject awakened by an alarm clock had high tensions. It is concluded that sleep per se, even deep sleep, may cause at most a trifling change in alveolar carbon dioxide tension, but that periods of very deep natural sleep occur when a mouth piece is not retained and the tension rises considerably.

BOOK REVIEWS

The Oxford Medicine by Various Authors. Volume VIII. Edited by Henry A. Christian, A.M., M.D., LL.D. Cloth. Loose-leaf. $25. Pp. 965. Oxford University Press, 114 Fifth Avenue, New York 11, 1953.

This book deals with the treatment and prevention of those diseases that are commonly seen by the general practitioner or the internist. Diagnostic and etiological data are omitted, because they are to be found in other volumes of this series. Treatments usually carried out by surgeons or other specialists have also been omitted. The book is bound with a flexible cover in a loose leaf binder so that individual chapters can be revised from time to time. Dr. Burgess Gordon states in the introduction that, "throughout the ages treatment has been the most difficult and unsatisfactory part of medicine," and points out that physicians having swung from empiricism to nihilism are now witnessing a great new era of specific chemotherapy. The authors have been conservative, including only those new modes of treatment that experience has proved effective. A short bibliography is included at the end of most chapters. It is noted with approval that leprosy is consistently referred to as Hansen's disease. In discussing amebiasis the authors appear to be a little overenthusiastic about emetine and the antibiotics, but this is a minor matter in a book that should prove an invaluable reference work for both student and practitioner. One source of annoyance is the use of paper that is thin enough to allow the print on the opposite side to show through. The index is excellent, and the book is highly recommended.

Films in Psychiatry, Psychology & Mental Health. By Adolf Nichtenhauser, M.D., Marie L. Coleman, and David S. Ruhe, M.D. Medical Audio-Visual Institute of Association of American Medical Colleges. Cloth. $6. Pp. 269, with illustrations. Health Education Council, 10 Downing Street, New York 14, 1953.

A distinct service has been performed to the field of mental health by publication of this volume. The text is preceded by a gallery of scenes from the films described. Professional persons, acquainted with the meaning of these sequences extracted from excellent educational films, will find them an incentive to further perusal of the book. A careful pruning of this material would have made the book even more valuable. For each film there is a general description, an indication of the audience for whom it is intended, production data and the name of the distributor, a description of the content, an appraisal of the presentation and effectiveness, and a section on utilization that elaborates on some of the previously covered points. The study includes the evaluation of 51 films under the sponsorship of the Association of American Medical Colleges and five foundation funds. Although the use of "psychiatrically oriented discussion leaders" is urged in most instances, the reader is never advised where such leadership might be procured. For example, the role of mental health associations and societies is omitted; there is no reference to state mental health authorities; the medical and paramedical staffs of inpatient and outpatient psychiatric facilities are not mentioned; and the authors have failed to acquaint their readers with the resources offered by psychiatrically oriented pediatricians, psychiatric social workers, school social workers, family casework and child care agencies, and specially trained educators in nursery schools for mentally retarded and trainable children. There is an abundance of "creative and talented discussion leaders," but there is a paucity of such persons with competence in the field of mental health. Persons with competence in mental health should be invited to discuss these films. In other words, the promotion of education via audiovisual aids has preceded the development of public understanding of professional services. This aspect of community education is overlooked by the authors.

The lumping together of such films as "Children's Emotions" and "The Steps of Age" with "The Feeling of Hostility" and "Out of True" is questioned. The volume, supposedly, is directed to all audiences. Not many persons in this universal group could distinguish between the superficial and the soundly based offerings. No member of the mental health team can fail to appreciate this pioneer effort to acquaint the field with detailed information on films that do or do not advance the understanding of human behavior with relation to mental health or mental illness. Perhaps a second or third attempt will result in the production of a better focused, more readable volume on the subject.

Method and Theory in Experimental Psychology. By Charles E. Osgood, Associate Professor of Psychology, University of Illinois, Urbana. Cloth. $14. Pp. 800, with 223 illustrations. Oxford University Press, 114 Fifth Ave., New York 11, 1953.

This valuable new reference book brings up-to-date the experimental work in human behavior. The author is eminently qualified for the task he has set himself, namely, to present "an evaluation of experimental literature in close relationship to modern theoretical issues." As a teacher and researcher in the field of experimental psychology for many years, he has contributed a number of noteworthy additions of his own, especially in the area of learning theory. The fact that the book was reviewed and criticized prior to publication by a number of recognized authorities has made it highly acceptable. In format and print it is especially good. It is not, as are many such books, overly difficult to read in terms of specialized psychological terminology. The author has stressed the newer area of symbolic processes, including language and communication, in a thought-provoking section. His own special field of learning theory receives a much needed critical evaluation, and the more frequently emphasized fields of sensory and perceptual processes are given adequate coverage. The list of references is exhaustive. Simply presented, easily readable, and well illustrated, this broad exposition of the experiments leading to an understanding of human behavior offers a useful reference volume. That part of the medical profession interested in the behavioral sciences should find it of extreme value and a worthwhile addition to their library facilities. To all those interested in present day experimental approaches to human behavior, the book should be a welcome addition to their resources on experimentation, classic research design, and modern theory in basic psychology.

History of the Second World War, United Kingdom Medical Series. Editor-in-Chief: Sir Arthur S. MacNalty, K.C.B., M.D., F.R.C.P. Medical Research. Edited by F. H. K. Green, C.B.E., M.D., F.R.C.P., and Major General Sir Gordon Covell, C.I.E., M.D., D.T.M. Cloth. £2. Pp. 387. Her Majesty's Stationery Office, P. O. Box 569, London, S.E.1, 1953.

This volume of the official medical history of World War II describes the research promoted by the medical research council and various government departments and also research promoted directly by the fighting services. While the work of the council was greatly expanded during those eventful years, there are some omissions from this volume, because certain subjects have been adequately covered in earlier volumes of the series. There is little or no mention here of wartime research in psychiatry or of wartime advances in anesthesia and in technical methods of surgery. Considerable similarity exists between the text of this volume and that of the report of the medical research council for 1939-1945, which was published in 1948 under the title "Medical Research in War." The chapters in this book include discussions of the safety and efficiency of the fighting man, wounds and injuries, war diseases (dyspepsia, tuberculosis, influenza, infectious hepatitis, serum jaundice, malaria, typhus fever, dysenteries, sprue, Q Fever, and poliomyelitis), nutrition and malnutrition, public health, sulfonamides and the development of penicillin, biochemical research,

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chemical defense research, and the biological effects of explosions. The chapter most interesting to practicing physicians probably is that on sulfonamides and the development of penicillin, in which there is a considerable history of the early research and clinical work of these revolutionary medical discoveries. The many references listed after each chapter are admittedly not complete and are almost entirely confined to British work; only a few publications from the United States and elsewhere are mentioned. Such omissions were intentional and understandable in a history of British medical research. As the editor-in-chief, Sir Arthur MacNalty, says, "Abundant evidence will be found in this volume to show how great were the advances made in medical sciences during this period."

Bailey's Text-Book of Histology. Revised by Philip E. Smith, Ph.D., Sc.D., and Wilfred M. Copenhaver, Ph.D., Professor of Anatomy, College of Physicians and Surgeons, Columbia University, New York. With assistance of Dorothy D. Johnson, Ph.D., Assistant Professor of Anatomy, College of Physicians and Surgeons, Columbia University. Thirteenth edition. Cloth. $9. Pp. 775, with 442 illustrations. Williams & Wilkins Company, Mount Royal and Guilford Aves., Baltimore 2, 1953.

A substantial amount of revision has resulted in this new edition of a classic textbook. There are nearly 100 new figures, many of them replacing older pictures but several, such as the electron micrographs, covering subject matter not included in earlier editions. The material in chapter 1 on the cell has been entirely rewritten to include recent advances in our knowledge of nucleic acids, enzymes, etc. Throughout the rest of the book, changes have been made to incorporate pertinent new information provided by the fields of histochemistry, electron micrography, and physiology. The more extensive use of bold-type headings improves the already clear and logical division of text matter. Reference lists at the end of each chapter have been brought up-to-date. The book does not pretend to be a source work for instructors or research investigators, although it covers the field more than adequately and is still one of the best histology texts available for medical and dental students.

The Management of Pain with Special Emphasis on the Use of Analgesic Block in Diagnosis, Prognosis, and Therapy. By John J. Bonica, M.D., Director, Department of Anesthesia, Tacoma General and Pierce County Hospitals, Tacoma, Washington. Cloth. $20. Pp. 1533, with 785 illustrations. Lea & Febiger, 600 S. Washington Sq., Philadelphia 6, 1953.

This book, which may serve as a textbook or a reference book, deals with the fundamental aspects of pain and the various methods of its management with special relation to analgesic blocks. The anatomic, physiological, and psychological aspects are ably discussed. Regional anesthesia is described in full detail with relation to drugs, doses, vasoconstrictors, armamentarium, and principles. This comprehensive book is well written and well illustrated. It should be in the library of every physician interested in anesthesia.

Sports Medicine. Edited by M. J. Karvonen, M.D., Ph.D. Proceedings of International Symposium of Medicine and Physiology of Sports and Athletics at Helsinki 17-18. 7. 1952. Paper. $3. Pp. 229, with illustrations. Suomen urheilulääkäriyhdistys, Finnish Association of Sports Medicine; distributed by Finnish Bookshop, Helsinki, 1953.

This work reports the results of an international symposium held in Helsinki just before the Olympic games in 1952. Nine of the contributions are in German, two in French, one in Spanish, and the rest in English. The contributions from Russia and other countries are presented in one or another of these four languages. The diversity of viewpoints and the inclusion of original data give the book a permanent documentary value. Some of the contributors, apparently ignorant of mathematics beyond the process of computing averages, do not seem to have known what to do with their material but deserve credit for making their figures available to other investigators. Of particular interest in this volume are the many passages (pages 18 to 24, 93 to 95, 116, 121 to 126, 145 to 154, 157, and 227) relating to the heart. Putting all the curious results together, one can only conclude that physicians who allow newspaper photographers to picture them applying a stethoscope over a pugilist's heart are making themselves ridiculous. Relating further to pugilism is a commendable study by a Finnish contributor of the electroencephalograms of seven former boxers and of two younger men with recent cerebral damage after boxing. This paper is followed by a vigorous discussion in English, which condemns boxing in words that every physician dealing with athletes should read. The editors and publishers have produced an outstanding work in languages not their own. It is recommended as containing something of value for every medical reader.

Lehrbuch der Geburtshilfe. Herausgegeben von Prof. Dr. Th. Koller, Direktor der Universitätsfrauenklinik in Basel, Prof. Dr. E. Held, Direktor der Universitätsfrauenklinik in Zürich, und Prof. Dr. W. Neuweiler, Direktor der Universitätsfrauenklinik in Bern. Bearbeitet von Prof. Dr. E. Held, et al. Band II, Lehrbuch der Geburtshilfe und Gynäkologie, herausgegeben von E. Anderes, H. Guggisberg, und Th. Koller. Second edition. Cloth. 62.40 Swiss francs. Pp. 907, with 348 illustrations. S. Karger, Holbeinstrasse 22, Basel, 1953.

The second edition of this book has been drastically reduced in size and is now printed in one volume instead of two. Much of the text and many of the illustrations had to be eliminated to accomplish this. The section dealing with fetal monsters is still too large and contains more than a fourth of all the illustrations in the book. The book is divided into 13 sections written by nine collaborators: E. Held, O. Kaeser, Th. Koller, A. Reist, H. Schultheiss, R. Wenner, A. Werthemann, H. Welli, and S. Wolf-Heidegger. The section subjects are (1) human embryology and morphology of the newborn, (2) normal pregnancy, (3) normal labor, (4) the puerperium, (5) physiology of the newborn, (6) multiple pregnancy and labor, (7) breech presentation, (8) pathology of pregnancy, (9) extrauterine gestation, (10) dystocia, (11) pathology of the puerperium, (12) fetal monsters, and (13) pathology of the newborn. Almost the entire field of obstetrics is covered, but there are a few omissions, such as amniotic fluid embolism and the lower nephron syndrome. All of the contributors are experienced obstetricians and teachers, and the information given about obstetric physiology and pathology is accurate. The advice given about the conduct of labor and obstetric complications is conservative and authoritative. In spite of the fact that nine men contributed to this book, the data are presented in a clear and uniform style. The illustrations are numerous, clear, and well reproduced. This volume should be even more popular than the first edition, because it is undoubtedly one of the best textbooks on obstetrics written in German.

The Allergic Patient and His World Including Sources of Allergens. By Florence Eastty Sammis, M.D., M.S.P.H., F.A.A.A., Assistant Attending Physician in Allergy, Nassau Hospital, Mineola, New York. Publication number 157, American Lecture Series, monograph in Bannerstone Division of American Lectures in Allergy. Edited by Max Samter, M.D., Chief, Allergy Clinic, University of Illinois College of Medicine, Chicago. Cloth. $4.75. Pp. 156, with illustrations by Oren C. Durham, Chief Botanist, Abbott Laboratories, North Chicago. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill.; Blackwell Scientific Publications, Ltd., 49 Broad St., Oxford, England; Ryerson Press, 299 Queen St., W., Toronto 2B, 1953.

This monograph is the best of the books available for the lay reader interested in allergy. The material has been carefully selected and clearly presented. The author has made good use of a wealth of clinical experience in writing this book for the layman. More than two-thirds of the volume is devoted to hay fever, asthma, food allergy, and allergy of the skin. The balance contains special instructions and recipes, a bibliography, a brief appendix that considers the question of vegetable oils as allergens, and a comprehensive index. The style is clear, literate, and concise. There is no attempt at sugar-coating the material with semi-humorous anecdotes or with unusual and frequently bizarre case reports commonly used to maintain reader interest. Interested lay persons will find answers to many questions: the results to be expected from hay fever or asthma therapy; the degree of relief; the possibility of cure; the value and limitations of corticotropin (ACTH), cortisone, and antihistaminics; how to prepare a dust-free room; and how to prepare a palatable restricted diet. In addition to being well

written, the book has been ably edited. The physician treating allergies may find it useful to have one or more copies available for circulation among his patients. The more informed such patients are, the more cooperative they will be in adhering to the difficult and prolonged treatment usually required.

Annual Review of Biochemistry. Volume 22. J. Murray Luck, editor, Hubert S. Loring and Gordon Mackinney, associate editors. Cloth. $6. Pp. 729, with illustrations. Annual Reviews, Inc., Stanford, California; H. K. Lewis & Co., Ltd., 136 Gower St., London, W.C.1; Maruzen Co., Ltd., 6, Tori-Nichome Nihonbashi, Tokyo, 1953.

Many reviews contained in the 1953 volume will interest specialists in nutrition. First, there is a chapter by A. G. Hogan of the University of Missouri entitled "Nutrition," which deals with 123 references on the subject of the relationship between maternal health and congenital malformation of offspring. Next is an account in the prefatory chapter by E. V. McCollum of his college training and of his first decade of research in foods and nutrition. Finally, there are chapters on the chemistry and biochemistry of carbohydrates, lipids, amino acids, proteins, nucleic acids, purines, pyrimidines, and vitamins. These chapters were prepared by outstanding men in their fields in England, Israel, Europe, and South America, as well as in the United States. Workers in other fields of medical science will welcome chapters on the biochemistry of enzymes, cortisone, neoplastic tissues, teeth, immunopolysaccharides, antibiotics, and vision. Of lesser interest to physicians are the reviews on nutrition of ruminants and on photosynthesis.

20 Years of Psychoanalysis: A Symposium in Celebration of the Twentieth Anniversary of the Chicago Institute for Psychoanalysis. Edited by Franz Alexander, M.D., and Helen Ross. Cloth. $3.75. Pp. 308. W. W. Norton & Company, Inc., 101 Fifth Ave., New York 3, 1953.

Several distinguished American physicians, educators, social scientists, and psychoanalysts have contributed to this volume celebrating the 20th anniversary of the Chicago Institute of Psychoanalysis. In his introductory review, Dr. Alexander shows how psychoanalysis has moved from its pioneering aggressiveness into a position of recognized responsibility. He cautions psychoanalysts by stating: "It requires that we abandon those defensive attitudes which developed at a time when psychoanalysis was emotionally rejected both by the public and by the academic world. I daresay that these defensive attitudes today are a greater obstacle than the emotional resistance of non-psychoanalysts." Dr. Alan Gregg contributes a stimulating essay, "The Place of Psychoanalysis in Medicine," focusing on the changing concepts of medicine, the changes in the practice of medicine, and how psychoanalysis can relate to these shifts. Dr. Martin Grotjahn considers new trends in psychoanalytic training; Dr. Lawrence Kubie writes on psychoanalysis as a basic science; Dr. I. A. Mirsky discusses psychoanalysis and the biological sciences; and Dr. Talcott Parsons considers psychoanalysis and the social sciences. There are also stimulating and pertinent discussions of each of these papers. Concluding sections describe the research, training, and publications of the Chicago Institute during this period. This volume is an excellent survey of the achievements, the orientation, and the challenges of American psychoanalysis.

Leistungssteigerung: Leistung, Übermüdung, Gesunderhaltung. Von Prof. Dr. Max Hochrein, Chefarzt der medizinischen Klinik Ludwigshafen/Rhein, und Doz. Dr. Irene Hochrein-Schleicher. Third edition. Cloth. 27 marks. Pp. 283, with 53 illustrations. Georg Thieme, Diemershaldenstrasse 47, (14a) Stuttgart O; [agents for U. S. A., Grune & Stratton, Inc., 381 Fourth Ave., New York 16], 1953.

The problem of increasing the output and efficiency of physical work in man is the subject of this book. The authors state their concept of *Leistung* on page 18 as the work effect that results from the synergism of psychointellectually conditioned drives and organically limited work capacity, two factors that vary widely and independently in each given case. The main body of the book deals largely with the nature and treatment of excessive fatigue and with procedures for improving health and efficiency. The book is valuable mainly as a digest of the references (about 600) listed in the bibliography, which is typographically accurate but incomplete and uncritical. It not only omits important contributions by such investigators as Cureton, Karpovich, Kleitman, and Schneider but also fails to indicate what the authors accept as proof for the assertions they quote from the literature. In a field where motivation is so important, the control of this variable is essential, yet there is little in the book to suggest that the authors appreciate the significance of a contratest or *Gegenversuch*. In the various recommendations for treatment, for instance that on page 235, they make no distinction between pure chemical substances and proprietary mixtures. According to a popular magazine, an American team of weight lifters went to a recent world championship meet in Stockholm with "a large case" of special tablets and powder. They "had vanilla, plain and chocolate flavors, with the plain being used by lifters who had to watch their weight." The temptation to ascribe successes to the use, and failures to the nonuse, of favorite preparations of this sort is irresistible, and the resultant vexing emotional conflicts need to be settled by systematic fact-finding rather than by the continued annotation and exegesis of past impressions and opinions. This book, which makes little headway in the right direction, can be recommended as a source of references for specialists in the field of work-physiology.

Basic Bacteriology: Its Biological and Chemical Background. By Carl Lamanna, Ph.D., Associate Professor of Bacteriology in Johns Hopkins University School of Hygiene and Public Health, Baltimore, and M. Frank Mallette, Ph.D., Associate Professor of Biochemistry in Johns Hopkins University School of Hygiene and Public Health. Cloth. $10. Pp. 677, with 100 illustrations. Williams & Wilkins Company, Mount Royal and Guilford Aves., Baltimore 2, 1953.

This is not an average or all-inclusive textbook of bacteriology dealing solely with the taxonomy of bacteria but rather a volume that considers the basic physiological and biochemical properties of micro-organisms. Thus the fundamental physical, chemical, and biological problems of bacteria are stressed constantly to emphasize the principles underlying various bacteriological phenomena. The result is a welcome departure from conventional texts, and many specialized phases of the sciences relating to the life processes of bacteria, are furnished in a clear, lucid style. This text is recommended primarily to students of bacteriology, but it should also prove of interest to general practitioners and interns.

Advances in Pediatrics. Volume VI. Editor: S. Z. Levine. Associate editors: John A. Anderson, et al. Cloth. $7.50. Pp. 323, with illustrations. Year Book Publishers, Inc., 200 E. Illinois St., Chicago 11; Interscience Publishers, Ltd., 2a Southampton Row, London, W.C.1, 1953.

This new volume brings together up-to-date authoritative information on seven subjects related to pediatrics. The emphasis in this volume is given to disorders of the newborn. The topics include preventive prenatal pediatrics; pulmonary pathology, hemolytic disease, and intestinal obstruction of the newborn; megaloblastic anemia of infancy; the lipoidoses; and lymphosarcoma in childhood. The detailed discussion of each of the subjects is prepared by a leading authority. Extensive bibliographic references are given. The intention is not to present new material but to sum up the present knowledge in certain fields in which rapid strides have been made in the past few years. The current volume also contains a listing of the subjects covered in each of the preceding volumes. A year-by-year collection of these volumes provides the pediatrician or general practitioner with complete up-to-date information on a variety of important subjects.

Handatlas der Cystoskopie und Urethrocystoskopie. Von Dr. med O. Kneise, a. o. Professor für Urologie an der Martin-Luther Universität Halle, und Dr. med. M. Stolze, Dozent für Urologie an der Martin-Luther-Universität Halle. Third edition. Cloth. 50 marks. Pp. 110, with 127 illustrations. Georg Thieme, Thomaskirchhof 21, Leipzig (10 b) C1, 1953.

This new edition of an atlas first published in 1907 has been enlarged and rearranged. It consists largely of a series of life-like water colors made of lesions in the bladder and urethra as seen through the cystoscope. As in the previous editions, this atlas has the advantage that the pictures were made by urologists who were also artists. The number of pictures visualizing the urethra and the bladder neck, unfortunately, is limited. The atlas should be helpful to the beginner in the use of the cystoscope and the urethroscope.

QUERIES AND MINOR NOTES

CONGENITAL BLINDNESS

To the Editor:—*A patient wishes information regarding the inheritance of blindness in the event of marriage. He is one of two brothers with congenital glaucoma. There is no other similar history in his family for at least three generations. The girl he intends to marry has congenital cataracts. There is also no similar history in her family.*

J. T. Bergmeyer, M.D., West New York, N. J.

Answer.—The question raised in this instance is a difficult human genetic prognostic problem. The occurrence of a sporadic case, the offspring of assumably normal parents, may result from three major causes: (1) expression of the homozygous state of a recessive gene, (2) a mutation occurring in the germ plasm of one of the two parents, and (3) a phenocopy, the sequela of noxious or infectious intrauterine environmental agents (toxoplasmosis, rubella, and other viruses). A thorough examination of the affected person and his available relatives, a careful pedigree, and meticulous maternal pregnancy history are all requisite before one is qualified to give genetic counsel. Both gene action and intrauterine infectious agents, singly or in combination, may act to effect developmental aberration of the anterior ocular segment and produce congenital glaucoma. A few of the gene-determined anomalies of the anterior chamber effecting congenital glaucoma consist of partial aniridia, hydrophthalmos, Axenfeld's syndrome, cornea plana, and varieties of microcornea. Each of these entities may exhibit different modes of hereditary transmission. Congenital cataracts, and the variety is not mentioned, likewise vary greatly in type and may also be the end-product of different genes or intrauterine environmental action.

With the data at hand, however, the following answer may be made: Both hydrophthalmos and congenital cataract may be genetic or nongenetic in origin. Assuming these entities to be genetic in origin in these two persons, then, because of negative family histories, they must be due to homozygosity (duplex state) of a recessive gene. So hypothecated, hydrophthalmos can occur among the offspring of the proposed marriage—in a ratio of 1 in 4 births—only if the woman with congenital cataracts happens also to be a carrier of the postulated rare recessive gene responsible for hydrophthalmos. Congenital cataract will appear among the offspring—again in the ratio of 1 in 4—only if the man with hydrophthalmos also carries the rare gene for congenital cataract. It is very unlikely that either of these persons is also a heterozygous (single gene state) carrier of the recessive gene responsible for the blindness of the other. It is not to be anticipated, therefore, that for genetic reasons the children of such a marriage will suffer from either of these two entities.

CHILD EATS TOOTHPASTE

To the Editor:—*Is there any toxic effect from the ingestion of chlorhydroxyphenyl (Baxin), which is used as a preservative in ammoniated dentifrices? Would any harm befall a 2-year-old child who ate half a tube of toothpaste containing this substance?*

Norman Bolker, M.D., Nampa, Idaho

Answer:—According to the manufacturer of dihydroxydichlordiphenylmethane (dichlorophene, G-4, Baxin), the acute oral toxicity of the material is as follows: D_{50} for guinea pigs, 1.25 gm. per kilogram of body weight; LD_{50} for dogs, 2.0 gm. per kilogram. Chronic toxicity studies on rats revealed that a daily intake of about 400 mg. per kilogram for 90 days was without effect detectable on gross or histopathological examination but that an intake of about 1 gm. per kilogram per day for the same period was associated with kidney changes.

The Council on Dental Therapeutics of the American Dental Association has information to the effect that the manufacture and distribution of the only dentifrice known to contain dichlorophene was discontinued in July, 1952. Before that, however, the product contained dichlorophene 0.25%, or 0.2 gm. in an entire 2.7 oz. tube of the paste. This quantity of the material would not be expected to produce serious effects if swallowed by a child weighing 10 kg. (22 lb.). Sensitivity reactions to dichlorophene have been noted in a number of persons and have been reported frequently in the literature (*J. Indiana M. A.* **42**:22-24 (Jan.) 1949; *A. M. A. Arch. Dermat. & Syph.* **64**:640-641 (Nov.) 1951; *New York J. Med.* **52**:1437-1438 (June 1) 1952; *J. A. M. A.* **151**:998-999 (March 21) 1953).

The answers here published have been prepared by competent authorities. They do not, however, represent the opinions of any official bodies unless specifically so stated in the reply. Anonymous communications and queries on postal cards cannot be answered. Every letter must contain the writer's name and address, but these will be omitted on request.

PERSISTENT POSITIVE SEROLOGY

To the Editor:—*A man of 25 with a history of venereal exposure was found to have a strongly positive blood test. He received 300,000 units of penicillin daily for 16 days. Three months later a test was negative. In another six months it was reported strongly positive. A second series of 20 daily injections of penicillin brought a negative report, but again it reverted to moderately positive in six months. A third course of penicillin was followed by a moderately positive report eight months after the last course of penicillin. His wife, whom he married after his first negative report, has a negative reaction now. What should be done?*

M.D., Ohio.

Answer.—A spinal fluid examination including cell count, quantitative protein estimation, quantitatively titered complement fixation test, and colloidal test should be obtained. If this is negative, no further treatment need be given. An indefinite continuation of antisyphilitic treatment wih penicillin, or by any other means, is not indicated for a persistently positive serologic test. Physician and patient should study the article by Moore, "Seroresistance (Wassermann-Fastness) in Syphilis: A Discussion for the Patient" (*Am. J. Syph., Gonor. & Ven. Dis.* **30**:125, 1946). It would be desirable to perform a treponemal immobilization test if facilities are locally available (see The Journal, March 14, 1953, page 962).

TRAUMA TO CHEST AND PNEUMONIA

To the Editor:—*A 9-year-old girl was hit on the left side of the chest by a softball on the school play ground the day before I saw her. The next day the chest over that area was sore. Examination revealed a hematoma over an area of 4 by 4 in. (10.16 by 10.16 cm.). There was no evidence of rib fracture; however, 36 to 48 hours after the injury, a high fever and pneumonia of the left lower lobe, with a leukocyte count of 25,000 per cubic millimeter, developed. The child responded to therapy with crystalline penicillin, and in a few days there was complete resolution of the process. It was my feeling that the trauma possibly precipitated the pneumonic process. The insurance company adjusters felt that trauma such as this, which did not cause rib fractures or penetrating wounds, could not possibly precipitate such a pneumonia. I would appreciate your comments.*

M.D., North Carolina.

Answer.—From the description of the injury and its cause, there scarcely seem to be any grounds for doubt that it was responsible for the pneumonic condition that developed. Regardless of any bacterial cause, a large variety of pathological changes can take place as a result of nonpenetrating trauma.

TRICHOMONAS VAGINALIS

TO THE EDITOR:—*I have worried over cases in which Trichomonas vaginalis are found not only in the vagina but also in bladder urine obtained by catheter and once in urine obtained directly from the kidney through a catheter. I have been successful in treating the vaginal type but can do nothing for the bladder and kidney infections. Can you suggest a treatment?* *M. Y. Dabney, M.D., Birmingham, Ala.*

ANSWER.—The role of Trichomonas vaginalis as an etiological agent in irritative infection of the vagina and urethra is very poorly understood. The organism is often found in persons who show no tissue reaction and who have no symptoms suggestive of an irritative factor. This is oftener true when it is found in the urine from the urethra or bladder than when it is found in vaginal smears. Some observers have felt that the Trichomonas organism in itself is nonpathogenic but that its action gives a specific Streptococcus a chance to become virulently active and to cause the inflammatory reaction. Trichomonas organisms are frequently seen in the prostatic strippings of a man who has no evidence of prostatic irritation or urethritis. Again, there is no question but that in other patients the Trichomonas infection has apparently been the cause of a marked irritation of the mucous membrane.

The original treatment of Trichomonas vaginalis infection of the urethra was irrigation with acriflavine, 1:1,000. Since that time, many other medicaments have been suggested. Some urologists have used silver nitrate in strengths from 0.1% up to 1% for topical application. Others have used merbromin (Mercurochrome) or thimerosal (Merthiolate) and mercurial derivatives. The best method of treatment of the urethra and bladder, if there is a direct connection, is to clear up the vaginitis. This is done by the usual prescribed methods, using a suppository for vaginal insertions twice daily and a daily thorough douche with an acid medium, the most available being a tablespoonful of vinegar to a quart of warm water. As is well known, Trichomonas vaginalis infection is a persistent disease, and it may be necessary to treat the large intestine with oxytetracycline (Terramycin), chlortetracycline (Aureomycin), or arsenical derivatives in order to destroy the focus of the organism.

ABSENCE OF SECONDARY SEX CHARACTERISTICS

TO THE EDITOR:—*What therapy should be given a 13-year-old girl who is physically small for her age and has shown no evidence of development of secondary sex characteristics. She is slim and somewhat shy but otherwise alert and of above normal intelligence. Could there be an agenesis of ovaries or hypo-ovarianism and how would these conditions be ruled out by laboratory methods? Would you advise estrogen and/or chorionic gonadotropin therapy for stimulating development of secondary sex characteristics? The parents are of average size and an older daughter is well developed and experienced the menarche at age 12.*

M.D., New York.

ANSWER.—A definite diagnosis is difficult in a girl of 13 years of age, as several conditions must be considered. She may have a retarded puberty without any abnormal endocrine function. Naturally, it will require a year or two to verify this situation. Other possibilities must be considered, however, such as ovarian agenesis in which there is a failure to grow properly associated with a lack of ovarian function. In these cases, pubic and axillary hair are usually present. Laboratory studies usually reveal an excess of gonadotropic hormone. If her growth has been stabilized for some time, this evidence would be considered indicative of dwarfism associated with ovarian agenesis. Another condition to be considered is a pituitary dysfunction in which there is a deficiency of the gonadotropic as well as the growth hormone from this organ. Although the diagnosis may be difficult in such cases, roentgenograms of the skull may be of value and perhaps a bone age study. There is usually an absence of gonadotropic hormone excretion. Treatment is not too satisfactory. The administration of estrogenic hormones may cause the secondary sex characteristics to develop, but the organs recede in size as soon as medication is discontinued. However, there may be some psychological value in this treatment.

DISCOID LUPUS ERYTHEMATOSUS

TO THE EDITOR:—*I have been treating a patient with discoid lupus erythematosus with quinacrine (Atabrine), for four months. The lesions have almost cleared, but I fear the development of some serious toxic state. Blood cell counts have been made without the discovery of any aplastic anemia or leukopenia. The patient has lost 6 lb. (2.7 kg.) in four months. Some diarrhea and vomiting have occurred. The highest dosage was 150 mg. daily, which was reached after two and one-half months. The dosage is now being slowly reduced in an effort to secure a small maintenance dose if possible.*

George M. Kesl, M.D., Port Huron, Mich.

ANSWER.—It would be inadvisable to alter the scheme of treatment in this instance. The lesions are almost cleared, there is no evidence of serious reaction, and the dosage is now being reduced to determine an effective minimum maintenance level. A better regimen than that has not yet been developed.

TOXICITY OF HYDROGEN SULFIDE GAS

TO THE EDITOR:—*Do hydrogen sulfide fumes have any effect on human lung tissue? A stream is highly polluted by organic waste from a large malting plant, and this gives off very offensive hydrogen sulfide fumes. At one time the fumes were so concentrated that they turned the lead in white paint on a home to a brown lead sulfate precipitate.*

M.D., Wisconsin.

ANSWER.—Hydrogen sulfide gas is exquisitely toxic, the upper limit of safety for extended exposure being not higher than 20 parts per million. Any available text dealing with occupational diseases or industrial hygiene will discuss this chemical. Hydrogen sulfide is both an asphyxiant and an irritant. When exposure is high, death may be brought about within a few minutes from central nervous system damage, and at autopsy, no tissue injury may be demonstrated. At the lowest level of damage eye irritation appears, and possibly this gas exercises selective action on ocular tissues. While lung involvement is not the rule, bronchitis and chemical bronchopneumonia have been clearly shown to have resulted. The offensive odor of hydrogen sulfide is no guide to prospective damage. Significant concentration quickly impairs odor perception so that the possibility is that so long as the odor may be detected, harmful concentration does not exist. White lead coatings readily are turned black because of the formation of lead sulfide, but this may be accomplished at such low levels that this coloration does not necessarily prove that bodily damage will appear.

TOEING IN AND TOEING OUT

TO THE EDITOR:—*I am often consulted about toeing in, toeing out, slight bowing in the legs, and knock-knees in children between 1 and 3 years old. Are there criteria that can determine what is and what is not within normal limits? When there is no actual deformity, to what extent is wedging of the shoe useful? Where are the heel and toe wedges placed? I have always recommended low shoes. Is this good doctrine?*

Frederic Speer, M.D., Kansas City, Kan.

ANSWER.—Toeing in may be due to (1) inward twisting of the tibia; (2) contracture of the internal rotators of the hip; (3) a change in the relation of the neck of the femur to the shaft; (4) contracted posterior tibial muscles; and (5) metatarsus varus. Toeing out may be due to (1) weak ankles and pronated feet; (2) loss of passive dorsiflexion of the foot when the knee is extended; (3) outward twisting of the tibia; (4) contractures of the soft parts about the hip; or (5) malalignment of the neck of the femur on the shaft.

In pronated feet, a lift should be placed under the heel, ⅛ to ¼ in. (0.32 to 0.64 cm.) thick on the inner side and a similar toe wedge on the inner side just under the great toe joint. With short heel cords, the heel of the shoe must be raised. When children who have toeing out are asked to stand on tiptoe, with short heel cords the foot goes into the corrected position. In metatarsus varus, the shoes should be worn on the wrong feet, that is the right shoe on the left and vice versa. In such cases, the wedges should be put on the inner side of the heel and sole so that, when the shoes are worn on the wrong feet, these wedges will be on the outer side. For the other conditions mentioned, the mother should be taught to give stretching exercises. Finally, inward twisting of the tibia will usually straighten out by itself.

CLEARING FACTOR IN PLASMA AFTER HEPARIN INJECTION

To the Editor:—*I read in a magazine that the National Heart Institute Group has discovered a "clearing factor" that serves in clarifying the blood of cloudiness after ingestion of a fatty meal. In other words this remedy is supposed to prevent accumulation of fatty substances in lining of arteries and thus help to prevent atherosclerosis. The remedy is supposed to consist of heparin and an enzyme extracted from animal stomach mucosa. Is this preparation of value? Is it obtainable and if so under what name and where?*

J. W. DeMand, M.D., Lincolnville, Kan.

Answer.—The studies mentioned by William Laurence in his article in *Look Magazine* are a portion of a general research program on the etiology of atherosclerosis. The "clearing factor" referred to is an enzyme in plasma that appears on intravenous heparin injection. Clearing factor activity also appears when animals are exposed to certain circumstances (e. g., peptone shock) that are known to release heparin into the circulation. The enzyme can also be produced in vitro by incubation of a precursor protein, present in plasma, with heparin in the presence of a tissue enzyme found in great abundance in pyloric mucosa. It can clarify lipemic plasma by catalyzing the splitting of neutral fat with the production of free fatty acids and glycerol. This splitting requires the simultaneous presence of serum albumin and a second material tentatively called "coprotein."

The over-all studies are still at the experimental stage and derive their clinical interest from the fact that the system will also attack the low-density lipoproteins earlier suggested as a possible etiological factor in atherogenesis. The clinical evaluation of the clearing factor system is under active investigation at the present time, but has not as yet led to any definitive therapeutically useful findings.

POSTOPERATIVE MENOPAUSE

To the Editor:—*Periodically a 30-year-old woman who had a hysterosalpingo-oophorectomy two years ago suffers from headaches, dizziness, nausea, nervousness, and depression associated with a rapid gain of weight and bloating. Although the severity and frequency of the occurrence of this group of symptoms are diminishing, they are disagreeable at times. There has been no history or indication of malignancy, and there are no mammary symptoms. What treatment will prevent or minimize these symptoms? Once the symptoms have developed, would estrogens aggravate or relieve the symptoms? What effect would the injection of an androgenic preparation have?*

C. L. Attaway, M.D., Ville Platte, La.

Answer.—This patient is typical of a rather large group of patients, and her symptoms are most probably menopausal. Estrogens often give partial or total relief. Most patients who have had surgical extirpation of all female pelvic organs do not have many symptoms after about two years. It is true the younger patients may have a harder time, especially if they are under stress. Most of these patients need little or no estrogen therapy. Sometimes giving estrogens intermittently prolongs the symptoms. Many of these patients have a combination of physical and psychic or emotional difficulties. Many are overweight, frustrated, and unhappy in their work or homes, and many are on poorly regulated diets. They should be examined carefully and closely questioned, allowing plenty of time for them to talk, relax, and "air" their problems. Then they should be put on well-balanced, high protein diets, which should also be designed for weight reduction if indicated. This often relieves the nausea and flatulence. Many of these women do well with administration of a little thyroid extract, depending on their thyroid function. A mild sedative such as phenobarbital in ¼ or ½ grain (15 or 30 mg.) doses may be prescribed, and the patient is told to keep them in the medicine cabinet in case she gets upset or cannot sleep. It is remarkable how many patients take none or very few of them. They are specifically instructed to eat three regular meals a day with no "piecing" in between. Adequate water intake is suggested for proper bowel regulation. Warm baths are suggested for relaxation, especially before retiring. Exercise, in the form of long walks, is advised, and hobbies are not only suggested but insisted on. These women should avoid situations that tend to make their symptoms worse. Lying down for a few minutes in the morning and afternoon tends to relax them. Usually by the time these routines are well established, the patient will "break down" and tell the physician the "real trouble"—her husband, her children, her boss, her co-worker—and often little suggestions correct the difficulty. In spite of the fact that most of the symptoms seem to be emotional, these patients should be observed often enough to make sure that no organic problem such as cholecystitis, gastric or bowel ulcer, or carcinoma exists. Much misunderstanding still exists concerning what happens following the removal of female pelvic organs, and before operation it should be explained to the patient so she will know what to expect. If the depression is severe and the above management fails to help, psychiatric care is suggested. Androgens are not superior to estrogens in therapy, and they often lead to masculinizing signs or symptoms.

CANCER OF BREAST

To the Editor:—*What is the present belief regarding the effectiveness of postoperative radiation therapy of carcinoma of breast in prevention of recurrence.*

P. W. Van Metre, M.D., Rockwell City, Iowa.

Answer.—The effectiveness of postoperative radiation therapy in the treatment of cancer of the breast is difficult to evaluate. Some studies indicate it is not of great value, while others claim from 5 to 15% improvement of five year survival figures by its use. Most surgeons employ it when the pathologist reports metastases to axillary nodes.

ANENCEPHALIC MONSTERS

To the Editor:—*In regard to the question of anencephalic monsters in Queries and Minor Notes in* The Journal, *Oct. 10, 1953, page 606, I am appending some of the published articles relating to the incidence of anencephaly. There appears to be a real difference in the incidence of this malformation in various countries.*

With respect to the question of how many pregnancies with anencephalic monsters go to term, data of MacMahon and McKeown (Brit. J. Social Med. **6**:*265, 1952) are pertinent. They reported that in a series of 473 such monsters, gestation was less than 33 weeks in 139 (29%), from 33 to 37 weeks in 193 (41%), and over 38 weeks in 141 (30%).*

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